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Quarterly Review of OPHTHALMOLOGY

Conrad Berens, M.D.
editor-in-chief

INTERNATIONAL RECORD

Editorial: Recent Advances in the Use of Antibiotics and Steroids in Ophthalmology
Conrad Berens, M.D., and Joseph Gulyash, M.D.

Diabetic Retinopathy
L. Benjamin Sheppard, M.D.

Abstracts on Military and Aviation Ophthalmology and Visual Sciences
Conrad Berens, M.D., F.A.C.S., and L. Benjamin Sheppard, M.D.

VOLUME 7 NO. 1 MARCH 1951

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INCORPORATING INTERNATIONAL RECORD OF OPHTHALMOLOGY

FOREWORD

The objective of the QUARTERLY REVIEW OF OPHTHALMOLOGY is to bring together in one publication concise but authoritative abstracts of current articles on ophthalmology, according to an all inclusive plan which will include all special, state, and national journals as well as the bulletins of clinics, hospitals, etc., and the transactions of meetings. This will embrace both the domestic and foreign literature.

To assist the reader to locate quickly the articles of current interest all data will be classified and published according to the following systematic plan:

Anatomy, Embryology, Heredity, Development and Nutrition
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Anomalies of Refraction and Accommodation, Contact Lenses
Conjunctiva
Cornea, Sclera and Tenon's Capsule
Anterior Chamber and Pupil
Uveal Tract and Sympathetic Ophthalmitis
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Vitreous Humor
Retina
Neuro-Ophthalmology, Optic Nerve, Visual Pathways, Centers and Visual Fields
Eyeballs, Exophthalmos and Enophthalmos
Glaucoma and Hypotony
Lacrimal Apparatus
Eyelids
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Hygiene, Prophylaxis, Occupational Ophthalmology and Injuries
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Miscellaneous
Book Reviews
Announcements

In each section there will be published a series of annotated references under the heading, "References to Current Articles." To save the reader's time and also to assist in the compilation of bibliographies these references will invariably refer to articles of an academic nature or those making similar reports to abstracts recently published on the same subject. A cumulative index in the concluding numbers of each volume will provide further aid in locating specific references. Constructive criticism will be appreciated.

A section entitled International Record of Ophthalmology is to be included at the beginning of the journal. The Record Section will consist of advanced clinical and experimental reports.

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Editorial Director and International Editor

Editorial Offices
19 East 62nd Street, New York 21, N. Y.

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667 Madison Avenue, New York 21, N. Y.

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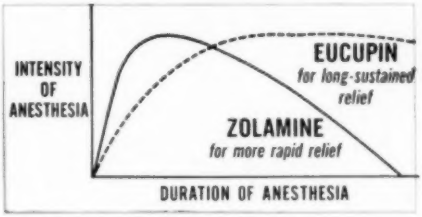

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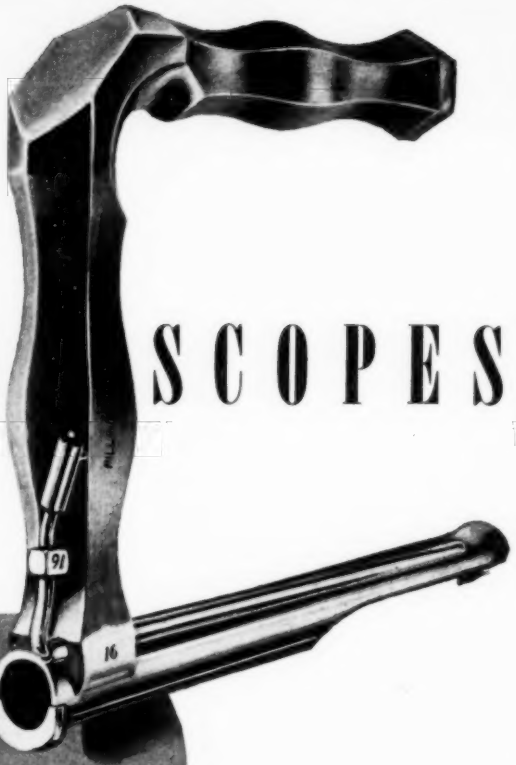

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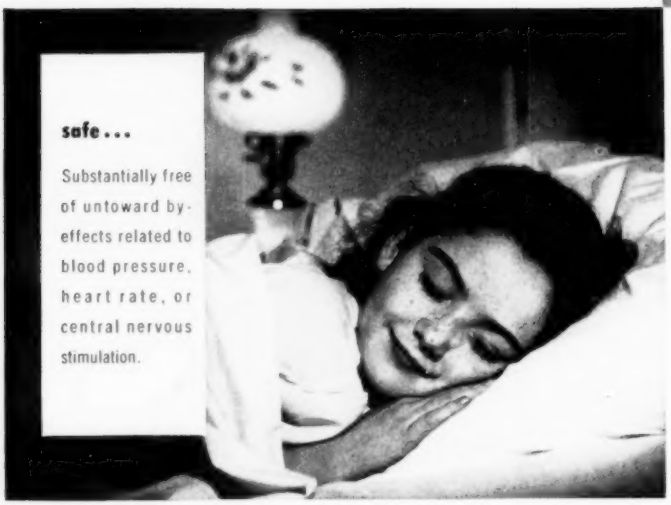

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Quarterly Review of **OPHTHALMOLOGY**

VOLUME 7 NO. 1 **MARCH 1951**

Incorporating the International Record of Ophthalmology

Recent Advances in the Use of Antibiotics and Steroids in Ophthalmology

EDITORIAL

Conrad Berens, M.D. and Joseph Gulyash, M.D.
NEW YORK, N. Y.

The newer concept of selective permeability of the cornea and sclera and the nature of the blood aqueous barrier makes it necessary to vary therapy of ocular infections as to the most efficient routes of administration, bacteriologic sensitivity, and toxicity of the drugs employed. The use of these drugs must be considered individually. Aureomycin, chloromycetin, dihydrostreptomycin combined with promizole or para-aminosalicylic acid, ACTH and cortisone acetate hold the spotlight in recent advances in ocular therapeutics.

Although the sulfonamides have been largely replaced by the antibiotics, sodium sulfacetimide in 30 per cent solution is still an excellent agent in treating certain types of conjunctivitis, corneal ulcers, and following minor injuries to the eye. Dunphy recommends its possible use as a prophylactic agent against ophthalmia neonatorum in place of silver nitrate.[1]

In the field of antibiotics, the antibacterial, antirickettsial, and antiviral potentialities of aureomycin and chloromycetin continue to gain favor among ophthalmologists in treating eye conditions.

Intolerable sensitivities, local or systemic, which are relatively frequent with the use of penicillin and the sulfa drugs, apparently are not so frequent with the use of aureomycin and chloromycetin. The successful use of aureomycin in treating brucellosis, tularemia, trachoma, and inclusion blennorrhea have been reported.[1] Its effect on herpes simplex, herpes zoster and epidemic keratoconjunctivitis is less favorable according to Braley and Dunphy.[1, 2]

Intra-ocular concentrations, using oral administration and topical application of aureomycin, are poor as compared with chloromycetin. Intravenous administration produces good intra-ocular concentration for both drugs. Neither drug can be given subconjunctivally because of considerable irritation. Detergents, iotophoresis, corneal abrasions, and corneal baths effect increased penetrability of antibiotics (penicillin, streptomycin, dihydrostreptomycin).[3]

The treatment of ocular tuberculosis, as reported by Woods and his co-workers, suggests the use of streptomycin or dihydrostreptomycin hydrochloride in conjunction with promizole.[4] Better results were obtained with a total dose of 42 Gm. of streptomycin and about 250 Gm. of promizole.[4] He suggests using 0.5 Gm. of streptomycin intramuscularly every 12 hours with 6 Gm. of promizole given orally in divided doses. Satisfactory clinical results are reported with a total of 26 Gm. of streptomycin combined with approximately 156 Gm. of promizole.

Bietti has used para-aminosalicylic acid successfully in the treatment of ocular tuberculosis, administering 20 Gm. orally in divided doses in combination with streptomycin.[5]

The Committee on Medical Research and Therapy of the American Trudeau Society has stated that dihydrostreptomycin has no advantage over streptomycin with regard to therapeutic efficiency, nor is it established that it is as effective as streptomycin.[6] However, vestibular disturbances were definitely less with the use of dihydrostreptomycin than with streptomycin.

Initial results with the use of ACTH and cortisone in ocular diseases are beginning to appear in the literature. The eye diseases in which ACTH and cortisone seem to have potentialities are the acute types of iridocyclitis: keratitis and scleritis.[7] Koff and his associates have reported the use of cortisone, administering 25 mg. per cc. subconjunctivally every 48-72 hours. Eight of 9 cases of iritis had remarkable improvement in the following sequence; first, pain disappeared within 12 hours; second, hyperemia of the eyeball decreased markedly; third, gelatinous exudate when present melted away in 24 hours; fourth, newly formed iris adhesions previously unresponsive to atropine were broken away and the pupil became widely dilated. Old organized synechiae were unaffected.[7] Encouraging results have been reported by Harvey, Howard, and Kattus in the treatment of sympathetic ophthalmia.[8]

Several cases of retrolental fibroplasia treated after the development of tortuosity of the retinal vessels, edema and early peripheral separation of the retina seemed to become arrested.[9] In chronic granulomatous types of uveitis, the use of cortisone or ACTH has not been encouraging.[6]

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Diabetic Retinopathy

L. Benjamin Sheppard, M.D.

DEPARTMENT OF OPHTHALMOLOGY, MEDICAL COLLEGE OF VIRGINIA, RICHMOND, VIRGINIA

During the past three decades many changes have taken place in the diagnosis and treatment of diabetes. The stimulating influence of the research laboratory, co-ordinated with the observations of clinicians, has evolved a natural change in medical thinking in regard to the diabetic. The purpose of this paper is to review briefly the fundamentals of this metabolic problem, with special reference to the ophthalmologic complications. The terms *retinitis, retinosis,* and *retinopathy,* as they apply to the retinal changes in the diabetic state, are not the subject of discussion. In this paper, we will speak only of retinopathy in the diabetic.

This review of diabetic retinopathy will summarize the literature and the findings relevant to an insidious, progressive, and degenerative metabolic disease, affecting all parts of the body. Every ophthalmologist has the responsibility of interpreting the eye findings in a diabetic so that the diagnostician may correlate them with his data in such a way that the patient may receive maximum benefit. In order to understand and interpret these findings properly, we must first have a working knowledge of how diabetes affects other parts of the body.

ETIOLOGY

Diabetes mellitus may be an hereditary disease.[81, 85] In John's[75] series of 500 diabetic children an hereditary factor was found in 31.5 per cent. Burnstein[20] presents observations on the anticipated hereditary tendency through five generations. Diabetes may also be a disturbance in metabolism due to various hormonal and neurovegetative influences. It is essentially a chronic disease, commonly developing in middle life, although diabetes occurs at all ages, even in infancy. Statistics[85] show there is an increasing chance that the nondiabetic may become diabetic as he approaches 50 years of age. This is especially true if he is overweight, for such individuals develop a poor tolerance for carbohydrates. The obese person at the onset of diabetes will usually have a tendency to develop an improved carbohydrate tolerance as he loses his weight and becomes a controlled diabetic.

The diagnosis is correct if there is a repeated positive urinary sugar, a fasting blood sugar above 129 mg. per cent, or a blood sugar above 164 mg. per cent following a meal. The preponderance of evidence shows that human diabetes is a pancreatic disease. The liver, the pituitary, the thyroid, and the adrenal glands may play an important part in addition to pancreatic factors. The importance of the adrenal cortical hormones and anterior pituitary hormones in the metabolism of carbohydrate, fat, and protein is best demonstrated by the experiments and discussions of Talbot[74] and Houssay,[72] which are too extensive for detailed consideration in this paper. Best[25] concisely defined diabetes as a disturbance of the carbohydrate, fat, and protein metabolism due to an imbalance of the glands of internal secretion. Diabetes has never been produced without injury to the pancreatic beta cells. The injury usually follows a period of great demands upon these cells, leading to exhaustion. Added to this may be a sudden shock (a toxic disturbance) for which the organ can no longer compensate.[127]

When man is exposed to disease or circumstances that upset his individual stability quotient, he may be said to be under stress or strain. His ability to readjust his physiologic processes to these adversities might be called individual resistance (or adaptability). It has been suspected for a long time (since Addison's classic description of adrenal cortical insufficiency in man) that the adrenal glands have an important function in the control of individual resistance to stress and strain. The anterior pituitary gland also plays a major fundamental role in the stress mechanism.[74] The body as a whole enters into the response to the stress, whether it is acute or chronic. Material pertaining to stress is found in the discussions by Selye[126] under "Adaptation Syndrome." During the stage of resistance following the "alarm reaction," most of the morphologic changes regress. The acquired degree of resistance to the stressful causative agent is at the expense of resistance to other types of stressful stimuli. If stress continues unabated, eventually there is exhaustion of the mechanism of resistance and the organism dies. Experimental data have been accumulated to show the important role of not only the adrenal cortex but also the entire related endocrine system in the stress mechanism.[139]

The present concept of pancreatic diabetes indicates that a continuously destructive agent, probably a toxic substance, causes gradual destruction of the islet cells. Warren[145] has stated that it makes no difference whether this is a toxic, hyaline, or hydropic change, it is a selective pancreatic destruction. At the same time that cells are being destroyed, other new cells are being formed, although they are not produced so rapidly as they are destroyed; therefore diabetes results and progresses. Allen[6] showed that the early hydropic degenerative change is a reversible process. Early diagnosis and control are therefore important.[108]

Today, there are approximately two million diabetics in the United States.[149] This large number is due to earlier diagnosis and to prolongation of life. Through education in the control of diabetes and various surveys, our population is becoming more conscious of diabetes. We are now able to discover diabetic cases sooner and encourage them to start with a specific therapy. This group is a public health problem as well as a medical one and certainly constitutes a fair percentage of a physician's practice.

Evidence has been presented that the basic underlying cause of ocular pathology in the diabetic is an altered metabolism.[29, 66] A brief review of both the related physiology and the research is in order. The blood sugar depends upon the intake and absorption of carbohydrates, proteins, and fats, and also upon the breakdown of body glycogen. The amount of exercise and the ability of the liver to store and release glycogen for metabolic needs also influence the blood sugar. The utilization of glucose depends upon the presence of insulin. The exact mechanism is unknown, although we know more about the action of insulin than that of any other internal secretion. Probably the first step in the utilization of glucose is its conversion to glucose-6-phosphate by means of the enzyme known as *hexokinase*. Insulin stimulates this enzyme reaction. The glucose-6-phosphate can be either converted into glycogen or utilized directly in the peripheral tissues. Cori[38] has shown that a hormone from the anterior pituitary inhibits the action of hexokinase, thus retarding the formation of glucose-6-phosphate. An adrenal cortical hormone may reinforce the inhibitory action of the anterior pituitary gland secretion.[132] Simpson's[130] case reports showed that when adrenal insufficiency is superimposed upon diabetes mellitus, there is a reduction of insulin requirements with an associated insulin sensitivity. Insulin over-

comes this inhibiting action of the anterior pituitary and adrenal cortical hormones, allows the formation of glucose-6-phosphate, and enhances its subsequent utilization.

The day has passed when we consider that glycosuria and hyperglycemia are always due to an insufficient supply of pancreatic insulin.[145] It is now apparent that dysfunction of several endocrine organs may account for the impaired carbohydrate metabolism, or the body requirements for insulin may be increased.[25, 45, 98]

The liver probably maintains a key position in the control of the body metabolism. The normal liver is adjusted to supply the amount of sugar required by the tissues and ceases production as soon as the blood sugar rises above this level.[132, 72, 40, 32] The uncontrolled diabetic uses carbohydrates at a hyperglycemic level, at the same time spilling large amounts of sugar, which points to the overproduction of glucose. The secondary changes associated with diabetes and the problems arising from disturbed metabolism are undoubtedly interrelated, not only in the field of endocrinology but also in that of nutrition.[91]

Associated with the lack of insulin, or an excess of an insulin-inhibiting agent, there is an overproduction of glucose from noncarbohydrate sources. Protein and fat are called upon to supply energy when carbohydrate is not available or cannot be utilized, as in the case of a total diabetic. In a total diabetic, no energy is derived from carbohydrates; about 30 per cent may be obtained from proteins and the remainder from fat reserves.

There are two ways in which fat may be used: (1) directly by the tissues, (2) indirectly by a partial breakdown in the liver to ketone bodies which are utilized by the tissues. In the absence of available carbohydrate, fat is broken down and acidosis results due to the overproduction of ketones. Briefly, insulin may be looked upon as a hormone somewhat similar to a catalytic agent, normally necessary for the proper utilization of carbohydrates, proteins, and fats. It is also necessary for storing an excess of carbodydrates, both as glycogen and as fat.

EYE FINDINGS

In diabetes we must first recognize that all the ocular abnormalities may not be due to the diabetic disease itself, for many are due to complications and associated processes.

The eye complications usually occur after the disease has progressed for 10 years or more. The exact time of onset of the disease is uncertain, as many patients have diabetes for an indefinite period prior to diagnosis. Since the large majority of diabetics now live longer, thanks to insulin and better care of the patient, we can expect to see an increase in the number of cases seen with associated eye pathology, especially in those poorly controlled. We can better understand some of the complications in the uncontrolled diabetic if we think of him as one who is overfed, yet undernourished. The patient is debilitated, consequently more susceptible to infections and various chronic disorders such as sinusitis, nephritis, tuberculosis, and arteriosclerosis.

Among the eye conditions observed are: wrinkling of Descemet's membrane, paralysis of accommodation, changes in refraction, cataract, disorders of the iris, optic neuritis, retinopathy, and glaucoma.

The wrinkling of Descemet's membrane, changes in refraction, and cataract are due to the altered amounts of salt and sugar in the ocular fluids associated with a disturbed metabolism. Added to these is an impaired nutrition, which gives a deficient nerve impulse, reduces the muscle tone, and thus causes an inadequate accommodation. The above process accounts for the fragility and hydropic changes in the iris pigment.[50]

The retinal findings are among the earliest pathologic changes found in the diabetic, so naturally they are frequently observed first by the ophthalmologist.

The statistical study of thousands of cases by Waite,[144] Van Hofe,[138] Cohen,[34] Lee,[94] Wagener,[142] and McKee[101] showed that approximately 23 per cent of the average diabetics had retinal changes, whereas they occurred in only 6 per cent of the nondiabetics. (Cohen 33 per cent, Waite 28 per cent, Von Hofe 10 per cent, Lee 31 per cent, Wagener 17.7 per cent, KcKee 20 per cent — average 23.3 per cent).

The physician must remember that severe uncontrolled diabetes may continue for a number of years without producing retinal changes. On the other hand, typical lesions may be seen in recently diagnosed diabetes, and these are the earliest warning signs of cardiovascular-renal degenerative disease.[20]

In 1890, Hirschberg[69, 70] classified retinal diabetic conditions as:

(1) Exudative forms with punctate central retinitis;

(2) Extensive hemorrhagic forms;

(3) A form with increased inflammatory signs, accompanied usually by renal insufficiency, with poor prognosis for vision and life.

Little has been added to the original classification.

Scattered minute hemorrhages and exudates, occurring most commonly at the posterior pole in a fundus that is otherwise normal, are generally considered diabetic in origin. These are usually located in the inner nuclear and outer plexiform layers with some extension to adjacent layers (Fig. 7). The earliest lesions are usually in the capillaries and venules. Several authors[53, 14, 66, 62] have pointed out that microcapillary aneurysms located in the inner nuclear layer are seen at an early stage, and on the first ophthalmoscopic examination may be confused with punctate hemorrhages. One differentiating characteristic of a diabetic hemorrhage is its long duration or slow absorption due to defective (or impaired) circulation. That these lesions may remain unchanged for a month or more points to the fact they are true aneurysms and not extravasations, which generally absorb in a few days[54] (fig. 1). Retinal petechiae are found in other conditions but usually absorb rapidly. In a diabetic, they persist over a long period, as Bedell[18] has shown by repeated fundus examinations and photography. In uncomplicated cases one is impressed by the absence of arteriosclerotic changes, retinal edema, and nerve changes. The smaller veins may be uneven with tortuosity. These findings may be seen early but usually are later observations associated with venous stasis, venous thrombosis, and hemorrhage.

The exudates are more frequently lipoid or cholesterol deposits, which are probably noninflammatory in origin (fig. 2). Diabetic exudates exhibit little tendency to coalesce. They are usually found in small clusters or as isolated areas, often with irregular borders. These areas are accumulations of yellowish white material, usually near the smaller vessels and capillaries. The exudates may form an incomplete ring (fig. 3). As recessions and recurrences take place, distinctive features of the pathologic development can be observed. The hemorrhages and exudates become more extensive as the disease progresses.

At the same time, there is dilatation of veins with nodular formations, occasionally venous loop formations and venous capillary proliferations[15, 106] (fig. 6). Occasionally, due to deposits of lipoid material in the vessel wall, localized areas of perivascular sheathing are found (figs. 4 and 6).

In the advanced stage there are huge hemorrhages associated with degenerative venous

sclerosis (phlebosclerosis), endothelial proliferation, and venous thrombosis. These hemorrhages may break through the internal limiting membrane into the vitreous, obstructing the view of the fundus (fig. 4). The retinal vessels are uneven in caliber and in some places attenuated. The characteristic punctate hemorrhages and exudates continue to be seen in the vicinity of the macula. Organization of the hemorrhage follows, leading to the formation of bands of yellowish white translucent scar tissue and new-formed vessels, a condition generally known as *retinitis proliferans*.

About 1.5 per cent of diabetics eventually have retinitis proliferans. It occurs in long-standing cases with progressive degenerative changes and in those poorly controlled. In this type of retinitis the sheets of connective tissue vary from the thinness of a delicate veil to the thickness of a heavy sheet. These partially cover and fan out from the optic nerve, especially above and temporally. At this stage there is definite interference with the ocular blood supply. The thin-walled (inadequate) new-formed vessels weave their way through the scar tissue from the central vessels (fig. 5). Bertha Klien[89] and Wagener[142] pointed out that there are two types of retinitis proliferans, the one mentioned above in which the formation of the connective tissue precedes that of the new vessels and a second type in which there is a primary formation of new-formed vessels, with or without evidence of a massive hemorrhage. Connective tissue proliferation follows.[139] These inadequate vessels rupture easily. In such cases the prognosis is poor. Many will not retain useful vision; some develop detachment of the retina (fig. 6) or secondary glaucoma. Occasionally, a huge choroidal detachment occurs, suggesting that vascular changes similar to those found in the retina may occur in the choroidal vessels, especially the choriocapillaris. However, these changes have not been found upon microscopic examinations of diabetic eyes.[54]

Microscopically, the thickening in the choroidal vessels has been demonstrated by Cohen.[24] There is often no visible direct relationship between the arteriosclerotic changes and the gravity of retinal changes. Many diabetic patients do not have visual complaints, although early hemorrhages and exudates may be found during the routine examination for refraction. It is wise, therefore, to study these patients under a mydriatic and search for evidence of early vascular damage. Retinal hemorrhages usually precede hypertension or vascular sclerosis in the arteries. Croom[39] found that hypertensive and arteriosclerotic changes in the retinal vessels were in most cases absent or minimal and therefore considered the vascular complications not inevitable.

When the average physician sees a patient with bilateral retinal changes at the posterior pole, he usually suspects that the condition is diabetic in origin. The main difficulties in diagnosis arise when there are complications of albuminuria, hypertension, and arteriosclerosis. It is true, as many authors[64, 37, 75, 41, 117] have pointed out, that diabetes existing for a period of from 10 to 20 years is frequently associated with a high incidence of vascular disease. Some hemorrhages and exudates in the retina will be absorbed. A few cases progress to complete blindness, owing to continued destruction of retinal tissue, by retinal hemorrhages and exudates, interference with blood supply, hemorrhages into the vitreous, scar tissue formation, and retinal detachment.[19, 64] Others may have associated vascular damage, as evidenced by nephritis, hypertension, and coronary, cerebral, or peripheral vascular disease. Our problem of differential diagnosis can be made easier if we will approach the diabetic with the assumption that he may also have other diseases.

As our population grows older and chronic diseases become more prevalent, our diagnosis must be keyed to an early recognition of the preclinical phase of the diabetic state.[108] More and more of the doctor's time must be devoted to the care or prevention of the degenerative diseases. Clinical and laboratory examinations must be directed more specifically to the study of the reserves of the various organs. Our whole population, thanks to the control of infections, lives longer. Diabetics, owing to the use of insulin, are attaining a greater age and it may well be that the aging of the diabetic population is a prime factor to be considered when complications arise. Mosenthal[103] has pointed out that, with advancing years and arteriosclerosis, the vascular changes commonly occurring in the diabetic may be unrelated to the diabetes *per se*, as they may occur in nondiabetic individuals. Arteriosclerosis is a progressive disease, affecting chiefly the peripheral blood vessels, especially those of the vital organs (heart, brain, kidneys), as well as the extremities. Louis Bauman[15] stated: "It is significant that retinitis and retinal hemorrhages are an almost invariable accompaniment of the foot condition and both are probably preventable." Careful early examination of the peripheral vascular bed may add information of value to both diabetics and nondiabetics.

The vascular findings in the fundus of a diabetic are frequently difficult to distinguish from those due to renal or arteriosclerotic disease.[24] It is by no means always possible to say from ophthalmologic study alone which of these three is present. Retinal edema, hemorrhages, and exudates occurring in a diabetic present a problem in differential diagnosis. A comparison of the edema in a nephritic with the hemorrhages, exudates, or vessel changes in a hypertensive and an arteriosclerotic will usually bring out the differences in the picture of a diabetic and that of a nondiabetic. The persistent deep punctate retinal hemorrhage is the most useful feature in the differential diagnosis of a puzzling case.

Wagener[141] reported that 60 per cent of diabetic cases showed retinal hemorrhages if they had had the disease over 10 years. In a series of childhood diabetics followed over 20 years, the frequency of retinal hemorrhages in those who had been kept under good control was one-fifth as great as it was in the group not well controlled.[118] Dolger,[41] in 1947, reported upon 200 patients followed for 25 years. All had retinal hemorrhages and many had albuminuria or hypertension.

In older reports lipemic retinitis was considered to be pathognomic of diabetes, but it is rarely seen at present. It is associated with extreme acidosis and may occur at any stage of uncontrolled diabetes. It is not a sign of vascular degeneration.

PROGNOSIS

The fundus picture does not always indicate the degree of severity of the diabetes, although it is the best index of progress. Joslin has pointed out that statistics are accumulated on diabetics who have died.[55] He has found that during the last 50 years 5 patients are living for every 1 that died with the onset of diabetes in childhood.[59] For all ages the duration of the diabetic life has risen from 4.9 to 14.1 years.[55] To prolong the life of patients is important, but it is more important for us as physicians to do all within our power to prevent disabling complications, which may manifest themselves as the disease progresses. Bailey[12] pointed out that obese, poorly controlled diabetics develop an associated progressive vascular disease. Jordan[77] emphasized that we can control the weight and blood sugar but not the duration. He felt that too much emphasis had been

placed upon the duration and not enough upon the average vascular changes expected with increasing age, whether the patient were diabetic or not.

Regardless of the duration of the diabetes, which so far is our only common denominator, the patients with no visible retinal changes are certainly in a preferred class in regard to prognosis.[66] Patients with punctate hemorrhages have a fair prognosis, but those with engorged veins have a tendency to perivasculitis or thrombosis, leading to massive vitreous hemorrhages, and their prognosis is poor.

The following conclusions are drawn from studying the reports on diabetic fundi:

1. There is an increase in the retinal changes, depending upon the duration of the diabetes, which is manifested chiefly by a disturbance of the smallest arterioles, capillaries, and veins.

2. A study of the fundus is the best index of the general progress of the patient.

3. The distressing fact is that once the pathologic process has started it usually progresses, although occasionally we see a retina clear after hemorrhages and exudates have occurred.

Recent studies have shown the importance of early fundus investigation in various vascular lesions. It is therefore necessary that the ophthalmologist, the clinician, and the pathologist adopt and use similar classifications and terminology, to insure mutual understanding.

PATHOGENESIS

We have not yet accumulated sufficient data to answer completely the questions how and why the metabolic disturbances in diabetes mellitus result in change in the blood vessels, with consequent nutritional disturbance in the retina. Some patients have retinal pathology without demonstrable vascular signs elsewhere in the body. Vulnerability can be observed better in the retinal vessels than in any other site in the vascular tree, for a small lesion can be seen there and may have far-reaching effects.

Frequent articles have appeared expressing various concepts regarding the etiologic background of the eye findings in the diabetic state. The following related phases of pathogenesis will be briefly presented: concepts, metabolic disturbance, hormonal relationship, relation of the kidney, vascular changes with particular reference to capillary insufficiency, and biochemical changes.

Through study of the occurrence of retinopathy in diabetes, two schools of thought have developed, representing these radically different concepts:

(1) That the hyperglycemia as such, whether controlled or not, is capable of producing the nutritive and secondary tissue changes, thereby accounting for the retinopathy;

(2) That the retinopathy is secondary to the vascular changes associated with the process of aging and shows favorable response to continuous control of the metabolic disturbance.

Dolger,[41] Tolstoi,[127] and Guest[65] have been the chief advocates of the first concept. Elwyn[47] and Fiegenbaum[50] suggest that an unknown toxic agent accounts for the capillary stasis and increased capillary permeability. Elwyn states that the retinal hemorrhages are a result of chronic capillary stasis. Definite findings are a slowing of the blood through the terminal vessels (due to a persistent hyperglycemia with a resulting capillary dilatation associated with anoxia) and impaired nutrition of the surrounding tissues, followed by

hemorrhages, lipoid deposits, and exudates. Elwyn[39] assumes that hyperglycemia is due to an inherited inferior central neural mechanism in the brain, probably in the hypothalamus; therefore, the stability of the normal blood sugar curve is not maintained. He considers that the pancreas is probably controlled through the vagus nerve. Braum[29] criticizes the proponents of the theory that disordered diabetic metabolism is the cause of retinopathy. He states that glycosuria and hyperglycemia do not cause the retinitis, since a similar finding is seen with hypoglycemia. Sindoni[131] expressed a similar opinion in stating that the true relationship of hyperglycemia and hypoglycemia to vascular degeneration is unknown. He felt that the slower-acting insulins may create a recurrent and prolonged hypoglycemia, with or without symptoms.

In the second concept, the vascular changes are primarily regarded as an accentuated aging process associated with hyperglycemia. Joslin[82] considered that the vascular bed of a diabetic is predisposed to presenility. Berens[22] gave a most comprehensive review of the eye changes in the aging process. He showed that the retinal vessels may have fatty globules in the intima and the media. The vascular degeneration may advance to the point of hyalinization of a vessel. According to Berens[22] and Rones,[116] impaired nutrition resulting from inadequate circulation is responsible for deposits of fatty globules in various structures.

In retinal vessels atheromatous changes may involve the larger vessels at the disk, but they do not occur in the smaller vessels; therefore they are not responsible for the retinopathy in the diabetic.[54, 145] More frequently we find thickening and hyaline changes in the media of the small retinal vessels. This, as shown by the work of Cohen[34] and Friedenwald,[55] causes an irregular lumen without complete obstruction and is not necessarily associated with hypertension. They pointed out that the changes may not always be seen ophthalmoscopically. Histologic studies of Cohen[34] and Friedenwald[55] also have proved that in the smallest arteries and arterioles, which have no elastic fiber layer, the primary indication of the arteriosclerotic process is a deposition of hyaline and lipid material beneath the endothelial layers in the basement membrane. This is a tissue response to disturbed nutrition, which is an indication of degenerative changes in diabetes. The fact that the onset of diabetes decreases in the later decades of life, the time when arteriosclerosis usually increases, is of significance.[83] This indicates that arteriosclerosis is not a cause of diabetes or of diabetic retinopathy.[143, 142, 54, 93] Subjective and objective manifestations of a disease arise as a result of effort on the part of an organ to regain its normal function by correction of the disturbed equilibrium. This process is retarded with aging. Cohen[33] considered that in diabetes the lesion was mainly vascular and therefore difficult to distinguish from arteriosclerotic changes. He concluded that we should consider the lesion an arteriosclerotic retinitis associated with diabetes. The hemorrages were due to degeneration of the terminal vessel wall and diapedesis. Cohen[34] found no venous stasis to explain the hemorrhages, contrary to Elwyn's concept. Gradle[63] made similar observations and considered that the retinitis was usually complicated by vascular and renal lesions.

Messinger[102] concluded that there was not enough evidence to support the assumption that bleeding from the smaller vessels in the diabetic is due to arteriosclerosis. Ricketts[111] pointed out that there are arterial changes in both arteriosclerosis and diabetes, but that only diabetes damages the capillaries of both the retina and the kidney.

Ballantyne[13] made a distinction between diabetic and hypertensive retinal lesions. In diabetes he found the lesion was primarily venous, while in hypertension the lesion was primarily on the arterial side. He considered the minute fatty granules in the vascular endothelium associated with a swelling of the endothelium to be the earliest diabetic changes, whereas the fatty granules in hypertension are more frequently found in the media. Scobee,[125] Agatston,[3, 4] and O'Brien[105] have been impressed by the importance of venous dilatation, the occurrence of which suggests that the earliest fundus picture in a diabetic may be dilatation of the venules and capillaries. Gibson[57] and O'Brien[105] have expressed the opinion that a number of diabetics, particularly those with mild long-standing diabetes, have sclerotic changes in the retinal veins. O'Brien assumed that these changes may be due to toxic and metabolic causes. These authors have demonstrated veins with hyaline changes, variations in thickness, and certain localized areas of constriction, while there may be other areas of thinning and dilatation through which components of blood cells may exude. Such findings, when they occur in the terminal vascular bed, may also explain the punctate hemorrhages and exudates. After the original capillary, venous, and endothelial changes, other developments may follow, such as an associated venous stasis, microcapillary aneurysms, phlebosclerosis, venous thrombosis, and hemorrhage. Recessions and recurrences may follow, sometimes leading to a complete disorganization of the eye.

The visible central retinal lesions are so much greater in number in a diabetic than they are in an arteriosclerotic that we must look for another explanation for the pathology in the fundus of a diabetic. Friedenwald[54] points out that, although the diabetic frequently has arteriosclerosis, the retinopathy is not a consequence of local sclerosis. McFarland[109] investigated the vascular changes of the eye associated with diabetes mellitus and concluded that the initial retinal changes occurred in the capillaries and the veins and that further progress probably depended upon the local vascular metabolic requirements of the tissue affected. The fact that diabetics are predisposed to infections is extremely suggestive of a vascular insufficiency, but there is not enough evidence to warrant the conclusion that diabetes promotes arterial or arteriosclerotic changes. The distribution of the vascular changes may be spotty, and this is an interesting field for research.

Ballantyne,[13] Loewenstein,[97] and Friedenwald[53] recently demonstrated that in a diabetic the capillary endothelium proliferates and encloses or partially encloses the retinal petechial hemorrhages. The authors assumed that in this process the original capillary leak or break may not be completely closed; therefore, repeated hemorrhages occur and a minute capillary saccular aneurysm may be formed by the proliferation of the endothelium. Friedenwald[55] assumed that the small capillary opening may not permit red cells to pass, although plasma proteins may escape, thus accounting for the deep, hard, round punctate exudative deposits. Ballantyne[13] suggested that the punctate white areas are thrombosed microaneurysms, and Elwyn[48] considered that the exudates follow deficient oxidation in the retinal tissue. The retinal hemorrhages and exudates frequently surround or are adjacent to the aneurysm, although they may occur in regions where there are no visible vascular abnormalities. They are usually found in the inner nuclear layer and the outer plexiform layer, and this suggests that there may be a leak from the capillary or the aneurysm. Friedenwald[53] has proved these areas to be true capillary aneurysms, using the Hotchkiss and McManus technic to demonstrate the basement membrane immediately beneath the endothelium. An afferent and an efferent connection with the capillary aneurysm are

shown. The demonstration of hyaline thickening of the basement membrane of the aneurysmal wall has been the most prominent feature, although hyaline thickening of the basement membrane may occur where there is no aneurysm.

Rodriguez and Root[113] reported that diabetic retinopathy seldom occurs in patients who do not have an increased capillary fragility, which is an early evidence of vascular disease. Beaser[17] had similar findings but found little improvement with vitamins C and P. Hanum[66] has shown increased capillary fragility in severe forms of diabetic retinopathy. More petechiae are produced by the tourniquet test in diabetics with retinopathy. Venous stasis and anoxia appear to aggravate the capillary instability.

If the capillary and the venule are the first focal points of a diabetic lesion, then the ophthalmologist has a most important place in directing the progress of the patient. The terminal vessels lying between the arterioles and the smaller veins are only endothelial tubes. They lie in the inner nuclear layer and may be looked upon as the terminal vascular system. The endothelium of this terminal vascular system may be easily injured, since it has a specific increased susceptibility, probably caused by toxins, anoxia, vitamin and mineral deficiencies, or substances produced by disturbed metabolism and endocrine imbalance. Degenerative vascular changes probably occur prematurely in the diabetic, especially in the capillaries and veins. There may be a venous subendothelial proliferation, causing a partial or complete venous obstruction. This may cause a slowing of the normal blood flow with an associated increase in capillary pressure, which may lead to capillary dilatation, a disturbance of normal functions, and a leakage through the vessel wall. It is logical to assume that repeated variations from hyperglycemia to hypoglycemia during relatively short intervals may predispose the capillary system to instability.

Hanum[66] reported that Dimmer, Osterlag, and Thannhauser experimentally produced retinal hemorrhages by causing wide fluctuations in the blood sugar with injections of insulin. This experiment would bear further investigation, for certainly the metabolic instability in the diabetic state may be a potent factor in producing the abnormal changes in the vascular and the nervous systems. Diabetes must cause an injury to the finer vessel walls, since the circulation is adversely influenced by the above-mentioned factors, and in many cases this is followed by the characteristic retinal lesion. The endothelial cells of the capillary wall may appear to be firmly united, yet they are normally capable of being separated to permit passage of white blood cells by diapedesis. Vascular complications have become more frequent, apparently depending upon the duration and the stability of diabetic control. Some evidence has been presented[75, 113, 80, 5] to show that prompt and continuous diabetic control does lessen the severity of the diabetes by allowing the early hydropic changes in the beta cells to return to normal. These encouraging findings have prompted a thorough dissemination of the facts about diabetes among our population, so that more cases are now being diagnosed and treated early. Senescence is certainly not the only factor that makes a diabetic susceptible to degenerative vascular disease.

The most important factors in the cause of retinitis proliferans in diabetics are the duration of the disease and inadequate control. Huge retinal hemorrhages usually precede the massive intra-ocular changes. The newly formed coils of vessels sweeping into the vitreous from the disk and connective tissue changes are nature's method of repair and organization, as shown in the works of Klien,[89] Hanum,[66] and Loewenstein.[97] Wagener[142]

stressed the importance of capillary proliferation, which is a response, to compensate for an impaired circulation in a slowly progressive degenerative disease. For the most part, these vessels are inadequate with soft walls and bleed easily. Gifford[58] reviewed the subject of proliferating retinopathy in diabetes and concluded that the larger veins were involved. Engorged veins associated with either partial or complete venous thrombosis may be the precipitating factor in the recurrent vitreous hemorrhages and the resulting formation of masses of connective tissue and new vessels.

Schneider[122, 123] showed that the plasma-albumin levels were lower than the beta globulin levels when the diabetes was poorly controlled and had an associated retinitis. Ruedemann's[120] clinical observations, reported in 1947, suggested that the lowered albumin content of the blood plasma may possibly be a contributing cause of retinal hemorrhages in diabetic cases. Best's[26] studies have shown the importance of the amount and nature of protein in our basal diet. Where there is sufficient protein to furnish methionine, there is no choline lack, except where there is interference in protein utilization. Schwartz[124] showed that the serum albumin can be returned to normal with an associated improved retinopathy, but it required a high protein diet over a long period. Research is now being conducted regarding the decrease of blood glutathione. Conn and his co-workers[36] have already shown that, experimentally, the intravenous use of glutathione caused a temporary drop in the blood sugar level in man when diabetes was temporarily produced with the pituitary adrenocortical hormone.

Hirsch and Weinhouse,[68] Duff,[43] Dragstedt,[42] and others,[145, 146, 67, 99] have presented evidence that atherosclerosis among diabetics may be due to disturbed fat metabolism. Wilens[147] found that atherosclerosis was more frequent in coronary arteries of the obese. Diabetics are frequently obese. One may assume that the intimal surfaces of arteries of obese persons are more continuously exposed to plasma rich in lipids than in the average person or one with poor nutrition. However, Steiner[134] found that intimal arteriosclerosis was not influenced by dietary cholesterol. The intimal lipid deposits have the same composition as the plasma lipids. The deposits in the tissue may simulate a foreign body or inflammatory reactions which are eventually reduced to fibrous plaques.

Once the progressive degenerative changes are started, they usually slowly progress with occasional remissions. Generally speaking, in degenerative diseases the blood lipids are increased, but in regenerative diseases the blood lipids are decreased. Dragstedt's[42] experiments with depancreatized dogs showed that there was a disturbed fat metabolism, which he felt was due to the lack of a lipotropic pancreatic secretion. He expressed the concept that arteriosclerosis as shown in the diabetic is a metabolic disorder. Associated with this idea is the fact that diabetics often have liver damage, poor fat metabolism, and a low prothrombin time. Duff[43] had similar findings.

Experimental evidence[51, 44] suggests that alterations in the blood cholesterol fractions may be an important factor in producing vascular changes in the intima of the larger vessels or in the media of the medium and smaller vessels, and subendothelial hyaline changes in the capillaries and venules. The latter is the primary vascular change in the diabetic. The capillary and venule offer the most probable site of pathology to explain the early retinal findings in a diabetic, which are frequently out of proportion to the arterial and renal damage. The evidence, however, is by no means conclusive. Biochemical studies may pave the way for the discovery of the cause of premature aging of the arteries

in a diabetic and thereby prolong his life. In turn, the data furnished will be of importance in throwing more light upon the field of degenerative vascular diseases. Cholesterol, whether free or esterified, if present as a protein-cholesterol complex, will be soluble in the plasma and probably can play no part in the production of the arterial lesions. Experimental evidence indicates that in normal plasma most of the cholesterol is in this form. There is evidence, however, that such is not the case in some conditions like diabetes, where fractions of the lipoprotein molecules may reflect the metabolic disturbance and develop atherosclerosis to a degree beyond that expected in supposedly normal individuals of a corresponding age.[69, 52]

Recent work by Holman[71] has shown that in dogs no arterial lesions were observed until renal function was disturbed. The author emphasized the importance of the "dietary factor," which is presumed to be lipid in nature. Best[26] and Soskin[132] found that a diet considered adequate in carbohydrates, fats, proteins, minerals, and vitamins, yet lacking choline or choline substances, will result in pronounced changes of function in the kidney and liver. These changes can be prevented in experimental animals by the addition of choline and methionine to the diet. These substances are called lipotropic agents, since they prevent fatty changes.

There is a similarity between the capillary findings of the eye and those of the kidney, in which the glomerular tufts permit the albumin to escape in cases of nephrosis. Where there has been a long-standing poorly controlled case, intercapillary glomerulosclerosis is a common diabetic complication and is associated with retinopathy, edema, hypertension, and albuminuria.[68] The works of Kimmelstiel and Wilson,[87] Wagener,[140] and Friedenwald[53] strongly suggest that changes in the capillaries of the retina and the kidney glomeruli are similar vascular processes specific to the diabetic. Allen[5] regarded the renal lesion as arising within the capillary wall.

Persistent capillary hemorrhages in the retina, which are probably capillary aneurysms, are considered characteristic signs of diabetes.[54] The intercapillary glomerulosclerosis in the glomerular tufts is also suggestive of diabetes.[87] Pathologists have concluded that the globular hyaline nodules near the periphery of the glomerules are the most characteristic lesions. The same type of hyaline material appears in the renal glomeruli and the walls of the retinal capillaries and capillary aneurysms. Ashton[11] and Friedenwald[54] have presented material showing a correlation between the retinal and the renal lesions. The evidence strongly suggests that they are identical lesions and manifestations of the same disturbed metabolic disease. Hanum[66] concluded that retinal changes in a diabetic develop even if there is normal kidney function. Any factor or factors seeming to give a satisfactory explanation of the pathogenesis of the diabetic vascular disease must show actual or potential capillary degenerative changes in the retina and glomerulus as well as in the remaining vascular tree. There must be two factors involved to produce the above changes:

(1) A disturbed or abnormal endothelium in the artery, capillary, or vein;

(2) A fraction of the blood lipids that is unstable and deposited in the vessel wall. To date, none of the experiments reported on cholesterol or hyperglycemia has brought out these two points.

Investigation of the metabolic fractions of protein,[136, 45] fats,[114] and carbohydrates,[67, 34] perhaps will lead us to facts explaining the vascular lesions specifically related to the

disturbed metabolism of a diabetic. By correcting or preventing the disturbing factors we may prevent, or at least retard, untimely progressive vascular changes in the diabetic.

Realizing our inadequate knowledge concerning the basic cause of diabetes has stimulated our interest in research in all branches of medicine. Goldner and Clark[61] found that man required less insulin and was more insulin-sensitive after total pancreatectomy. Dragstedt's[42] work on dogs agrees with the above. The questions arise whether a man with diabetes would be benefited over a long period by total pancreactectomy and whether he could be controlled with an improved long-acting insulin.

The observations of Cori et al[107] and Young[151] show the inhibiting effect of anterior pituitary extract on hexokinase activity with cessation of this inhibition upon the addition of insulin. The responsible substances appear to be closely related, although at present their exact identity has not been established. It appears that the diabetogenic activity of prolonged treatment with impure anterior pituitary extracts depends upon an exhaustion of Langerhans islets. At first there is stimulation with a period of hyperplasia, but as injection is repeated in the dog, there is destruction of the pancreatic islets.[126] Diabetes is being studied from an endocrinologic standpoint, as it is caused by a specific hormone deficiency. Soskin[132] has illustrated the endocrine relationship.

Many advances and improvements have been made in insulin since its discovery. It is stable and specific in its action, but it has many side effects which may be overcome with recent modifications of protamine zinc insulin. A newer type of insulin, known as NPH insulin, has recently been put on the market. This acts slightly more rapidly than PZI. We know little of the secondary action of insulin on proteins and fats. The dream of a treated diabetic, whether he has suffered one or more complications or not, would be to have an insulin that would become more active as the blood sugar rises above the normal range, with decreasing activity as the blood sugar falls below it. Some that dream awake refreshed with new ideas, new enthusiasm, and renewed imagination that may lead to more research with the result that he and his fellow-man may live to enjoy a more productive life. Ant[10] and Ricketts[112] have made interesting observations which indicate that protamine zinc insulin is not an "ideal" insulin for all. Insulin relieves the disease but does not retard degenerative vascular changes. The "ideal" insulin, now hoped for, will prevent *all* the complications of diabetes, enabling an insulin-treated individual to make the same contribution to society as the insulin-secreting individual.[25] Is it possible that some protective factor is present in the normal beta cell hormone that is lacking in the artificially prepared hormone? The answer to this question remains for the future, although our accumulated statistics comparing the vascular changes in the diabetic and the nondiabetic suggest an affirmative answer.

The study of the vascular complications associated with the diabetic state is our most urgent challenge today. If inadequate control is responsible for the degenerative vascular changes, then newer insulins and stricter diabetic regimens may be the answer to the challenge. If, on the other hand, the vascular findings are an integral part of the disease and not related to the defects in metabolism, then strict diabetic control would be of little consequence.

The evidence given earlier in this discussion tends to establish the etiology of diabetic retinopathy as a vascular lesion at the capillary level. This pathologic condition is intimately associated with a disturbed metabolism. The search for the specific factor or factors

FIG. 1. *Early Diabetic Vascular Changes.* Small dark punctuate hemorrhages in a fundus that is otherwise normal.

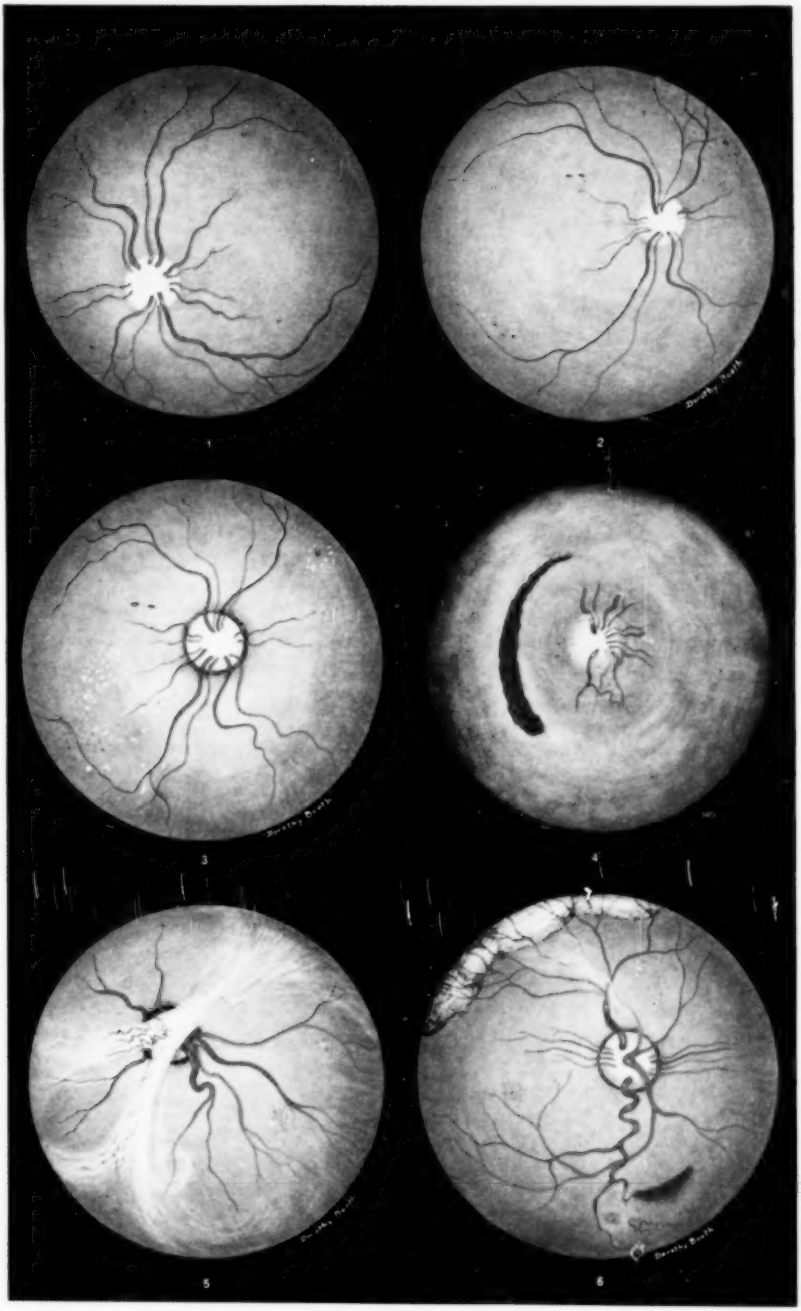
FIG. 2. *Diabetic Retinopathy, Mild.* Minute hemorrhages and yellowish white exudates scattered at posterior pole. Uneven veins.

FIG. 3. *Diabetic Retinopathy, Moderate.* Persistent punctate hemorrhages. Increase in the exudates, which tend to form a partial ring at the posterior pole.

FIG. 4. *Diabetic Retinopathy, Advanced.* Vitreous hemorrhage, severe, with clouding of vitreous, so that only nerve head and surrounding sclerotic vessels can be seen.

FIG. 5. *Diabetic Retinopathy, Advanced.* (Retinitis proliferans with and without new-formed vessels.) Thin bands of translucent scar tissue fanning out over fundus nerve head, with interlacing new-formed vessels. Torsion of veins and perivasculitis.

FIG. 6. *Diabetic Retinopathy, Advanced.* Torsion and sacculation of veins. New-formed vessels, retinal edema. Preretinal hemorrhages. Uneven caliber of vessels, smaller branch attenuated. Retinal detachment.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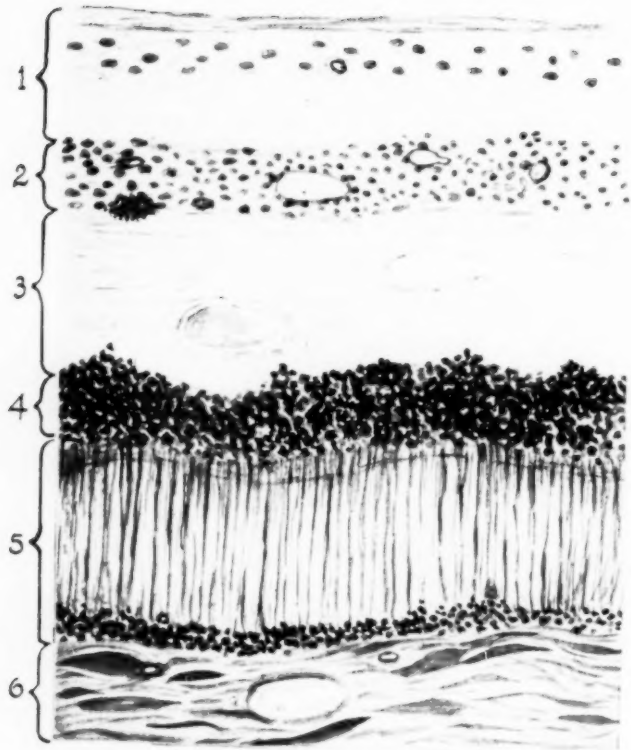

FIG. 7. *Diabetic Retinopathy. Cross section of Retina and Choroid.* 2. Inner Nuclear Layer. Left third—Hemorrhage (dark area); Middle third—Exudate (cystic space); Right third—dilated capillaries. 3. Outer Plexiform Layer. Large Exudates (cystic space). 6. Choroid—Thin-walled vessels.

causing this vascular damage is constantly being conducted in many fields related to diabetes.

Recent experimental studies offer new channels of hope. Alloxan,[21, 47] a chemical with a selective specific action on the islets (beta cells) in the pancreas, produces diabetes in 100 per cent of animal experiments. We look hopefully toward a day when some new therapeutic measures may avoid this destruction of the beta cells in the early stages of diabetes and may restore or stimulate the normal pancreatic functions. Experimentally this is now possible. By the use of atomic isotropes as radioactive tracers, the mysteries of growth are being unraveled. Perhaps a similar method soon will be devised that will bring us a step nearer to complete understanding of the function of the beta cells and the hormone insulin.

When retinal complications arise, we look for arterial and venous changes or increased capillary permeability. Often we find no cause for such developments and come to the conclusion that most likely a combination of factors is operating to produce the picture of a vascular disease found most frequently in the retina or kidney of a diabetic. The search for a satisfactory explanation of the fundus picture remains a definite and constant challenge to clinical and experimental investigators.

PATHOLOGY

Various organs and tissues of the body are involved in the diabetic state. Most authors agree that diabetes does contribute varying degrees of damage to the nerves, skin, or cardiovascular system. Autopsy material shows that arteriosclerotic changes are slightly higher in the diabetic than in unselected cases.[96, 147] The atheromatous type of change in the large and the medium-sized vessels is definitely increased. Deaths since the use of insulin seldom occur from coma, but 70 per cent are due to cardiovascular renal disease.[96, 112] Newburger and Peters[104] observed in their clinical and postmortem examinations of diabetics intercapillary glomerulosclerosis and retinal vascular changes. They are of the opinion that diabetes, hypertension, and renal involvement were associated with, or secondary to, extensive arterial and arteriolar degeneration. Colwell[35] concluded that, even though the late features of edema, albuminuria, hypertension, and hypoproteinemia were not present, the appearance of hemorrhagic or exudative retinopathy in diabetes must lead to a strong suspicion of glomerulosclerosis in the early stage. When x-ray evidence of arteriosclerosis is found in the legs of diabetics of 10 years' duration, there is apt to be associated coronary pathology. From the histopathologic findings there is sufficient evidence to assume that the retinal lesions are due to a primary or secondary vascular affection. The initial lesion is probably in the smallest vessels at the capillary level. This lesion is probably associated with the interrelated metabolic disturbances and not with the kidney dysfunction, arteriosclerosis, or hypertension. The author states that the efficiency of the liver function appears to be a factor in premature development of arteriosclerosis in diabetes mellitus. He goes on to suggest that impaired liver function may influence the normal utilization of fats and proteins, allowing a higher content of cholesterol and nitrogenous products, which may also aggravate the arteriosclerotic changes.[109] No difference has been found in the type of arteriosclerosis in the diabetic and the nondiabetic.[96] However, the sclerotic changes show a high incidence in the diabetic at an earlier age than is usually expected in the nondiabetic. This supports Joslin's contention

that the diabetes is a factor in predisposing the diabetic to sclerotic changes.[55] Recent evidence suggests that an elevated cholesterol often accompanies early arteriosclerosis.[123] To quote from Joslin,[55] "Arteriosclerosis is a sequel rather than an antecedent of diabetes and this opinion was strengthened by the demonstration that the incidence of onset of diabetes began to decrease in the sixth decade." Joslin[55] also strikingly states that the diabetic who in the preinsulin era died of acidosis now lives to die of coronary disease or gangrene. Diabetes was listed as the twenty-seventh most important cause of death in the United States in 1900, but in 1949 it was in the seventh place.

We urgently need more material in early diabetes so that additional tissue studies can be made.[54] The large group of diabetics in our practices and clinics furnishes an opportunity to study the process of aging and associated degenerative changes as well as being a source of material necessary for these studies. Ascertaining the cause of these presenile findings involves also a study of the endocrine and biochemical factors. How they influence metabolism is our greatest problem at present, and a challenge for future investigation. Through these investigations, the exact cause of the condition may be found, and we can then concentrate our therapeutic forces more directly upon this problem. It is, therefore, necessary to have the close collaboration of the biochemist, internist, pathologist, physiologist, and ophthalmologist, that together we may systematically describe and classify the differentiating ophthalmologic characteristics of diabetic retinopathy, with its associated clinical findings, and demonstrate this distinction from the findings in the hypertensive or nephritic type.

TREATMENT

To obtain the best results with diabetes, as with any other disease, one must conservatively handle the patient's case as individual and not merely use a routine treatment. The chief problem is to have the patient find and secure an internist who knows diabetics and how to care for their problems.

A good basic knowledge of general medicine is as valuable to the opthalmologist as it is to the internist if fundus findings are to be correctly interpreted in their relation to an internal disease.

In 1922, Joslin[79] said: "One by one, the mysteries are dropping out of the treatment of diabetes." Prior to the discovery of insulin in 1921, we made a great advance in the treatment of diabetes when undernutrition therapy was introduced in 1914. *Undernutrition* means a fasting diet.

Baumann,[16] in 1921, concluded that the keynote of a proper method of treatment was undernutrition. He advised carbohydrates to the tolerance limit and then the addition of protein. We still believe in the insulin era that a diabetic should not be overfed. He cannot utilize food nor can he replace tissue loss due to fasting as quickly as the normal individual. The same is true when tissue is lost by infection or disease.

For the diabetic, insulin has been the most wonderful therapeutic discovery of this century. It has allowed him more freedom of diet, of work, and of travel, as well as a prospect of living many useful years. His life span is now more than three times as long as before. Statistics show that 85 per cent of diabetic patients take insulin.

Two distinct schools of thought have developed with regard to diabetic control. One, represented by rigid standards and controls, was set forth by Allen[8] and Boyd[27] and modified by Joslin.[84] This school presents the ideal control of the blood sugar and glycosuria

through the 24 hours. For best results, control must extend over long periods, using a corrective diet with or without insulin.[27] Boyd states: "No halfway measures of adequacy of treatment may justifiably be considered in comparing various regimens of therapy."[28] In a later review[27] he found that carefully controlled patients have fewer complications. The ideal objective of such control would be to restore the individual to normal. Few patients attain or even approach this goal. After many years of practice, Allen concluded that the complications of a diabetic are uniformly preventable and, if present, may generally be arrested by vigorous control.[6] Rabinowitch[108] presented evidence that the use of free diets was definitely harmful. The fact that many diabetics who have had the disease over 20 years have been poorly controlled has led to the allegation that diabetics have complications regardless of control. Many authors[15, 7, 85, 27, 46, 118] have reported that patients who have maintained satisfactory control over long periods have fewer vascular degenerative changes.[110]

Lawrence[90] believes that many diabetics with eye lesions improved under treatment. This and other reports[6, 78] offer a gleam of hope to the diabetic who conscientiously follows his physician's instructions. The largest series found so far is 2,002 diabetics reported by Waite and Beetham.[144] In their opinion, "deep retinal hemorrhages in diabetics multiply with age and duration of diabetes out of proportion to the age factor, but they show no obvious correlation with sclerosis of retinal vessels, with vascular hypertension, with renal disorders, with insulin dosage, or with blood sugar and calcium levels."[143] Dolger[41] concluded that the duration of diabetes was more important than the method of treatment or the degree of control. Conn[37] and Griffith[64] came to essentially the same conclusions in regard to the ocular vascular manifestations. The retinopathy bears a definite relation to the duration of the diabetes, but it is related less clearly to the postdiabetic regimen of the individual patient.[27] Many of the patients referred to by Dolger,[41] White,[146] and in a J.A.M.A. editorial,[66] who have been diabetics for 20 to 25 years, undoubtedly lived at a time when we considered it wise to keep carbohydrates low and make up the calories with a high fat diet, which is the exact antithesis of what we do today. This diet led to poor results when compared with what we get today with a relatively high carbohydrate, high protein, and relatively low fat diet. We cannot be certain, but it is possible that the high fat diet may have had something to do with the increased incidence of vascular pathology.

The other school presents a quite different point of view. This school maintains that the occurrence of sugar in the urine is of no concern, especially in diabetics treated with protamine zinc insulin, provided there is liberal carbohydrate intake and insulin is given in sufficient amounts to prevent acidosis. The reports by Tolstoi[137] and Guest[65] demonstrate the advantages of a free choice of food, under supervision. The aim of diabetic control should be to attain and maintain as nearly as possible normal physiology. Persistent sugar in the urine or an elevated blood sugar is abnormal. Perhaps an inbetween course may be most desirable in treating an elderly patient with retinopathy. Many authors advocate a mild glycosuria in an elderly arteriosclerotic diabetic.[59, 24, 73] A slightly elevated blood sugar guards against possible insulin shock and perhaps is the lesser of two evils in an individual with fragile blood vessels.

Diabetic control is difficult to define because of the complicated metabolic ramifications. The lack of agreement among specialists as to what constitutes effective control certainly

adds to the confusion of the average physician, not to mention the bewildered state of mind of the patient.

For many years Joslin[55] and Allen[7] have led the way in increasing longevity for diabetics by advocating a strict control. Lee[95] has stressed the practical importance of maintaining general measures designed to retard aging and stimulate repair. With this in mind, the use of high-protein diets with the addition of vitamins and minerals has been advocated for both the prevention and treatment of diabetic retinopathy.[124, 31] Williams[150] arrived at similar conclusions and stressed the importance of adequate carbohydrate intake and maintaining a nitrogen balance.

Sindoni[131] has pointed out that the control considered adequate in general practice, based upon a 24 hour period urinary sugar and a fasting blood sugar, is not sufficient. Such control has not decreased materially the incidence of vascular degenerative complications. It is better to determine how a patient can utilize the usual meal by examining the urine and blood sugar two to three hours after that meal.

It may be true that the use of insulin has not materially decreased the frequency of degenerative vascular lesions, but it has postponed them. The effectiveness of insulin in the control of a diabetic is shown in the mortality statistics.[53]

When we assume that the control of the diabetes alone will solve the problem of diabetic retinopathy, we are probably overlooking other equally important factors.[146] Associated with a disturbance in the utilization of carbohydrates[8] are disturbed fat metabolism,[9] disturbed protein metabolism,[150, 45] and disturbed fluid and electrolytic balance.[73] It is quite possible that additional factors affect certain individuals and that diabetes renders blood vessels more susceptible to toxins, either metabolic[133] or bacterial. The search for, and the removal of, existing foci of infection often give dramatic results and prevent retinopathy in the diabetic.[23, 24] Capillary fragility and prothrombin time should be routine determinations for diabetics. There seems to be a possibility that many retinal hemorrhages and certainly many cases of retinitis proliferans may be prevented by early diagnosis of retinopathy and treatment of cases of increased capillary fragility with rutin and ascorbic acid.[59, 76, 129, 115, 63, 119] If the prothrombin time is found prolonged, vitamin K should be given. Ascorbic acid and rutin should be given at least three times daily. The two are most effective if administered simultaneously. The petechial index and the intraocular pathology will improve in more than half of the cases.[128] However, recurrences and recessions may occur in spite of continued treatment. Rodriguez and Root[115] found that with increased capillary fragility, retinopathy and nephritis often developed and were evidence of general peripheral vascular disease. The same authors found that increased capillary fragility was related more closely to the duration of the diabetes than to the age of the patient. This finding agrees with the clinical findings of Messinger[102] and Dolger.[41]

The term *adequate control* is not definite, but the following objectives should be considered:

(1) To enable the patient to maintain physical and mental well-being and at the same time hold an even weight;

(2) To maintain the fasting blood sugar and blood sugar levels two to three hours after meals within a reasonably normal range, without wide fluctuations in the blood sugar during the 24 hour period;

(3) To prevent polyuria and keep the urine free from sugar as far as possible. Fluid intake is important to prevent dehydration and assist in nitrogen elimination;

(4) To keep the albumin-globulin ratio within normal limits;

(5) To maintain a normal blood cholesterol;

(6) To maintain normal blood pressure and normal renal function.

The first visit to the doctor's office is of the greatest importance. The history (personal and family) and both physical and laboratory examinations are a prime necessity to the physician; without them he cannot give adequate instructions to the patient in the regulation of his diabetes. The patient must realize the gravity of his disease without losing the hopefulness that is acquired through proper education in the care of one's self.[54]

Insulin, if required, should be started at once in small doses and gradually increased or decreased as required. Younger diabetics usually require regular crystalline insulin in addition to a slower-acting insulin. Middle-aged diabetics can usually be controlled with protamine zinc insulin. Older diabetics may learn to control themselves with diet alone. It is better for the patient to learn how to use one type of insulin wisely and certainly not more than two. The physician should not change the type of insulin without an excellent reason.

The patient should be told that insulin is being given him to replace its deficiency in his own body, and that it may be necessary to increase or decrease this from time to time, according to his varying needs. Infections or injuries will usually make it necessary to increase the insulin. He must fully understand that insulin makes possible nutritionally adequate diets and that it will control his disease. Severe cases require large amounts of insulin; therefore the chances are greater for variations from hyperglycemia to hypoglycemia. The stricter the standard of treatment adopted, the more necessary are the care, training, and follow-up of the patient.

If possible, a new diabetic should first be admitted to the hospital for the initial studies and regulation of the insulin. While there, he should be allowed to go into a mild insulin shock before being discharged. He should experience the feeling of marked nervous instability that precedes the fade-out. He must realize the potency and the danger of this new drug. He has to learn how to counteract an insulin reaction and has to be made to realize that a lump of sugar in the pocket, when used at the right time, may spell the difference between life and death. The intelligent and cooperative patient will soon learn to proportion his diet to his requirements and activities. Frequent small meals, instead of the usual three, may give a better utilization of the food, may counteract shock, and prevent hyperglycemia. The quality of the diet is of the greatest importance. Individual requirements vary, but carbohydrates from 150 to 200 Gm. and proteins from 75 to 150 Gm. are generally sufficient for the average daily activities of an adult. The fat intake should be limited, although the average portions suggested above will necessarily include fat. The amount of fat allowed is determined by the control of body weight. Diet should be selected with special reference to the necessary minerals, amino acids, and vitamins. Time and experience will give us better diet modifications as well as improvements in our insulin. When a patient takes an unusual amount of exercise, additional carbohydrates and proteins are required to furnish the necessary calories. The lack of these may cause the mild diabetic to go into insulin shock or the severe diabetic may burn excessive fat and go into acidosis. Instill into the mind of every diabetic the concept that diabetes requires a system

of living with one's self in his own home. He must be well schooled in self-discipline in order to maintain a constant body harmony. The use of common sense is the first line of defense. Constant vigilance in the regulation of diet, insulin, exercise, and personal hygiene constitutes the second line of defense. The proper control of diabetes from day to day is a patient's own responsibility.[83] Joslin has expressed exceedingly well the ideal doctor's responsibility in the following quotation: "Return visits at prescribed intervals are important—whenever the patient comes back, it is up to the doctor to tell him something new about diabetes so he will have confidence that the doctor is seeking to learn how to improve his care."[84]

Our aim should be to help a patient continue to be active and useful in his or her community endeavors and thereby retain a feeling of security. Careful and continuous supervision cannot be overstressed. Regular visits to the physician are important in helping the patient to keep in satisfactory balance, his diet, weight, exercise, and insulin. Such visits may also help the patient to prevent infections and avoid wide fluctuations in blood sugar. A physician should seek to further the patient's welfare by a sympathetic understanding of his or her whole condition as well as the immediate complaint. A physician's duty is to give the patient an objective, a definite task to perform that will stimulate hope and peace of mind. Such an objective may be to inspire the diabetic to adhere strictly to his routine and thereby compete with the nondiabetic for longevity. As Joslin so admirably says: "There should be a doctor-patient partnership."[80] The advantage of such a situation is that the patient is always in contact with the doctor and the disease will be better controlled, for the patient will be disposed to report any unusual signs or symptoms. The control of a diabetic may also be conditioned by certain psychologic mechanisms. Diabetics out of control sometimes develop psychic states.

Most of our larger clinics have a diabetic department. The patient's education and counseling in these clinics constitute the foundation of a diabetic clinic. Adequate instruction by physicians, nurses, and dieticians is most important. This can be accomplished for large groups in the out-patient departments. With the shortage of hospital beds and the increased hospital expense to a patient, more and more people will find it necessary to seek out-patient or office care. There must be close cooperation with the social service department. In recent years, diabetic education has been promoted by diabetic lay groups and the staff members of diabetic homes and camps. The association with other controlled diabetics helps the individual in character-building, upon which his physical improvement may largely depend. The well informed diabetic will promptly adjust his diet and insulin to handle trivial complaints but will recognize important complications and consult his physician, thus preventing serious difficulties.

Diet and insulin are not in the true sense a cure for diabetes. They represent a palliative method rather than a restoration of the affected organs to the normal. Most diets are intended to conserve body protein and prevent degenerative changes. Experience has proved that adequate carbohydrate utilization will spare endogenous proteins from destruction. Williams[150] suggests that the metabolic factor may also have an influence upon fluid balance. Glycosuria will lead to a destruction of body protein and a decrease in the available liver glycogen. Both of these can be controlled by the use of insulin. Adlersberg[2] recommended high-protein low-fat diets in the treatment of mild and moderate diabetes. Protein has a slower absorption and liberation of carbohydrate. Diets high in

protein are indicated in an insulin-treated diabetic with liver damage, nephrotic syndrome, or other forms of nutritional failure. In a discussion of renal complications in young diabetics of over 12 years' duration Adams[1] advised a strict treatment during the early years of the disease to avert the insidious complications of the renal syndrome.

COMPLICATIONS

When a diabetic has no complications, it is wise to advise him of his good fortune and to discuss with him the seriousness of the dangers of poor control. In any case, it seems worthwhile to give continuous control a trial for an extended period. The end justifies the means and is a challenge to the patient. No patient can be promised that he will not have, with his diabetes, complications such as retinopathy, peripheral neuritis, nephritis, cardiac thrombosis, and gangrene. The ability of the individual to regulate his diabetic state so that the blood sugar will continuously remain within relatively normal limits is the best index of control.[6] Patients that maintain such control will have fewer vascular lesions.[146]

The principal causes of the upsets of a diabetic are accidents, infections, and disregard for the balance in diet, insulin, and exercise routine. Infections are largely controlled by chemotherapy. Of the complications associated with imbalance, acute hypoglycemia is most serious. Hypoglycemia is a possible danger with protamine zinc insulin, which acts for longer periods than 24 hours,[131] and may cause changes in the central nervous system and blood vessels. Sindoni[131] observed that hypoglycemia associated with the slower-acting insulins may contribute to vascular degeneration in a manner similar to the effect of oxygen deficiency. This appears to be a reasonable assumption when we consider the fundus findings in diabetic retinopathy. The findings of Braum[29] substantiate the above. Diabetic coma is a rare observation with our present-day therapy. Prompt and heroic measures are required to restore a physiologic balance in this serious complication.

Most physicians have noted a definite decrease in gangrene of the extremities since the use of protamine zinc insulin. Mosenthal[103] ascribed this fact to the patient's being under constant insulin control throughout the 24 hour period, thereby diminishing the wide excursions of the blood sugar when the patient is not under control or when using only regular insulin, which acts for four to five hours. For the same reason, the vascular eye lesions may be ultimately less frequent. When we have advanced our scientific control to the point where we can prevent wide fluctuations in the blood sugar through a delicate balance of food, insulin, and exercise, then we shall have arrived at a place where most of our vascular and neurologic complications will be eliminated. For most patients this will mean a control day by day, year after year; for some, a possible restoration to the normal.

Our chief aim in diabetic therapy is to prevent the early complications. Once the vascular degenerative process becomes manifest, it is difficult to reverse, although it may be retarded.

The present-day treatment of diabetics is good, but it can be improved with an anticipation of better results. Further research is needed on the vascular changes in the diabetic state, with special emphasis upon:

(1) Nutrition and disease and how they affect the body;

(2) Heredity;

(3) Improved insulin and insulin administration;

(4) The endocrine system with respect to the interrelated functions;

(5) The interrelation of carbohydrate, fat, and protein metabolism;

(6) Electrolyte and water balance;

(7) Correlation of the above studies with further investigation of degenerative vascular changes in the fundi, and their relation to the associated changes in other organs, especially those changes due to disturbed metabolism.

A solution of the problems which manifest themselves in the eyes of diabetics may lie in a more intensive consideration of the above-mentioned factors.

SUMMARY

Review of the literature concerning the retinopathy found in the diabetic suggests:

1. Early and regular ophthalmoscopic examinations are important.

2. There is evidence that the primary retinal lesion is an abnormality of the capillary bed and a deposit of lipoid and hyaline material beneath the endothelium of the smaller vessels.

3. The duration of the disease is important, but constant individual control is the most important single factor.

4. Prophylaxis is at present our only safeguard against retinal diabetic complications. Uncontrolled diabetes produces nutritive and secondary tissue changes.

5. In many older diabetics there is more than a coincidental relationship between the degenerative vascular changes which manifest themselves in the eye and the kidney.

6. The present review of the literature on diabetic retinopathy suggests that we should consider that the retinal pathologic findings are influenced or precipitated by a number of factors other then hyperglycemia.

7. The explanations so far presented fail to account for all the retinal changes involved in the diabetic state. Any suggested etiology cannot be too rigid, must necessarily have limitations and overlap other possible retinal vascular changes, but as such it may be of value in the attack upon the problem through clinical and experimental studies.

Through these investigations the exact cause of the condition may be found, and we can then concentrate our therapeutic forces more directly upon this problem. It is, therefore, necessary to have the close collaboration of the biochemist, internist, pathologist, physiologist, and ophthalmologist.

CONCLUSION

1. The clinical and experimental observations as regards diabetes mellitus are reviewed.

2. The possible causative factors are genetic, hormonal, and dietetic.

3. Suggestions are presented to aid the ophthalmologist in the evaluation of the fundus, so that the patient and the internist may be advised, and to give a background for these suggestions.

4. Known related fields of research are reviewed and the study of other possible fields suggested.

ACKNOWLEDGMENT

My appreciation is extended to Dr. William R. Jordan, Dr. Isabelle Taliaferro, and Dr. J. S. Forbes, who have willingly cooperated with me as the presentation of this subject progressed.

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Abstracts

on

Military and Aviation Ophthalmology

and

Visual Sciences

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Foreword

VOLUME I

It has been said that "literature is a very bad crutch but a very good walking stick." This volume of abstracts of the literature on Military and Aviation Ophthalmology through the year 1940 should prove to be an excellent "walking stick" for the ophthalmologist, specialist and all those interested, practically or scientifically, in vision and ocular functions.

For everyone charged with the duty of safeguarding the pilot and his passenger, these data are invaluable because the eyes are the most important physical factor in the maintenance and flying of an airplane. The early beginnings of aviation ophthalmology and the gradual acquisition of ophthalmologic knowledge concerning the airplane and the pilot are described.

There is no phase of aviation medicine that has not progressed through the years. It can be said also that there is no phase of aviation medicine that cannot progress in the years to come. Even the flight surgeon and military surgeon in an isolated field, camp or ship may have such important data concerning past and present practice in military and aviation ophthalmology to guide him. To aid this group, abstracts concerning certain eye diseases and disorders of function are included. Research workers in the visual sciences and especially the scientists working on selection, training and care of aviators and military personnel will have completely cross-indexed material at their fingertips.

A perusal of this book indicates clearly that there was a lapse of interest in military and aviation ophthalmology between World War I and World War II. This does not mean that civilian flying did not progress, but research on military and aviation medicine lagged and discoveries were not immediately put to practical use. An illustration of this point is the fact that x-ray technicians used red goggles for many years, but night flyers only adopted them during the second World War.

The constant rapid changes in warfare, improvements in the airplane, and its increased speed have required constant vigilance on the part of engineers, physicians, and military personnel, as well as civilian authorities entrusted with the welfare and efficiency of our military forces.

A reference work of this type is an aid to all interested in the visual sciences and military and aviation ophthalmology, whether in a laboratory, operating room, camp, air field, battleship, or submarine.

R. W. Bliss,
Major General,
The Surgeon General, USA

Preface

The increasing importance of the eye in aviation and other military services has stimulated the compilation of this bibliography with selected abstracts. The work was undertaken with the encouragement and support of Major General David N. W. Grant, the former Air Surgeon, and the financial aid of the Ophthalmological Foundation, Inc., and The John and Mary R. Markle Foundation, Inc.

Although military and aviation ophthalmology naturally includes the entire field of ophthalmology, the only general references which have been included concern the diagnoses and methods of treatment of certain eye diseases and eye injuries. However, an attempt has been made to compile all references on advances in military ophthalmology and closely related subjects through the year 1940. Standard procedures described in textbooks of ophthalmology have not been listed but reviews of certain textbooks have been included.

The preparation of the abstracts has been facilitated by the assistance of: Dr. Michael J. O'Connor, Dr. Olga Sitchevska, Dr. Bruno Priestley, Mr. R. P. Edward Hopf. However, the abstracts could not have been compiled without the continuous painstaking effort and assistance of Mrs. Helen F. Sells.

In compiling this bibliography the following volumes have been consulted: (1) Hoff, E. C. and Fulton, J. F.: A bibliography of aviation medicine, Springfield, Illinois, and Baltimore, Md., C. C. Thomas, 1942; (2) Hoff, P. M., Hoff, E. C. and Fulton, J. F.: A bibliography of aviation medicine—supplement, published by the Committee on aviation medicine, National Research Council, Washington, D. C., 1944; (3) Quarterly Cumulative Index Medicus from 1925 to 1940; (4) The Journal of Aviation Medicine from 1930 through 1940; (5) Index-Catalogue of the Library of the Surgeon General's Office; (6) Abstracts of Aviation Psychology, Indexed, edited and typed by the Psychological Index of the Works Progress Administration, Civil Aeronautics Authority, Department of Commerce, September 29, 1941; (7) Bibliography of Aeronautics, Part II Medicine, U. S. Works Progress Administration, 1937; (8) McFarland, R. A.: Bibliography on the selection, training and physical fitness of aviation pilots, Washington, 1939; (9) Psychological Abstracts.

Dr. Louis H. Bauer, editor of the Journal of Aviation Medicine, has graciously granted permission for the inclusion of abstracts appearing in the Journal.

The American Medical Association has also granted permission to use Russian abstracts appearing in the Archives of Ophthalmology.

The author is grateful to the publisher, the Washington Institute of Medicine, for excellent cooperation in the preparation of this work.

CONRAD BERENS, M.D.

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E. Foreign bodies in eyeball
F. General considerations of eye injuries
G. Glare
H. Glasses
I. Lens
J. Light
K. Muscle balance
L. Optic nerve
M. Orbit
N. Parachutes
O. Retina
P. Therapeutics (see therapeutics—injuries)

LII. Lacrimal apparatus
A. Altitude (see altitude-anoxia—lacrimal apparatus)
B. Acceleration—deceleration
C. Diseases

LIII. Landing

LIV. Lens
A. Altitude (see altitude-anoxia—lens)

LV. Light sense
A. Altitude (see altitude-anoxia—light sense)
B. Colored lenses (see goggles and eye glasses)
C. Dark adaptation
1. Altitude (see altitude-anoxia—light sense)
2. Age
3. Anoxia (see altitude-anoxia—light sense)
4. Effect of drugs (see drugs—dark adaptation)
5. Examination (see examination—light sense)
6. Night Flying
a. Fatigue (see fatigue—night flying)
7. Tests (see examination—light sense)
8. Vitamin A deficiency
D. Dazzling
E. Examination (see examination—light sense)
F. Glare
1. Goggles (see goggles and eye glasses—glare)
2. Injuries (see injuries—glare)
3. Sunlight
G. Malingering (see malingering)
H. Miscellaneous data
I. Night vision
1. Malingering (see malingering—night blindness)
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LVI. Malingering
A. Examination (see also examination—malingering)
B. Night blindness

LVII. Medicine, Aviation, general consideration of

LVIII. Monocular vision

LIX. Muscle balance
A. Altitude (see altitude-anoxia—muscle balance)

B. Binocular fixation
1. Altitude (see altitude-anoxia—muscle balance and altitude-anoxia—field of binocular single vision and binocular fixation)
C. Convergence
1. Altitude (see altitude-anoxia—near point of convergence)
D. Cyclophoria
E. Depth perception
F. Disqualifying defects (see disqualifying defects)
G. Equilibrium of the ocular muscles
H. Examination (see examination—muscle balance)
I. Fatigue (see fatigue—muscle balance)
J. General discussion
K. Heterophoria
L. Hyperphoria
M. Injuries (see injuries—muscle balance)
N. Monocular vision (see monocular vision)
O. Orthoptic training (see orthoptic training)
P. Physical therapy (see orthoptic training)
Q. Refraction (see refraction)
R. Stereopsis (see muscle balance—depth perception)
S. Tobacco (see tobacco—muscles)
T. Treatment (see therapeutics—muscle imbalances and orthoptic training)
U. Sighting eye
LX. Neuropsychiatry (see psychiatry)
LXI. Nutrition
LXII. Nystagmus
A. Altitude (see altitude-anoxia—nystagmus and altitude-anoxia—equilibrium)
B. Equilibrium (see equilibrium)
LXIII. Oculocardiac reflex
A. Examination (see examination—oculocardiac reflex)
LXIV. Operational fatigue (see fatigue—flying stress)
LXV. Ophthalmoscopy
A. Examination (see examination—ophthalmoscopy)
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LXIX. Physiology
A. Altitude (see altitude-anoxia—physiology)
B. Physiology of vision
LXX. Psychiatry
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A. Visual reaction time (see visual reaction time)
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B. Contact lenses (see contact lenses)
C. Examination (see examination—refraction)
LXXVI. Rehabilitation
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LXXVIII. Research (see also special subjects)
LXXIX. Retina
- A. Acceleration (see acceleration-deceleration—retinal blood pressure)
- B. Altitude (see altitude-anoxia—retina)
- C. Injuries (see injuries—retina)
- D. Tobacco (see tobacco—retina)

LXXX. Safety in aviation
LXXXI. Spatial orientation
LXXXII. Standards, visual
- A. Australia
- B. Belgium
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- D. China
- E. Civilian aviation
- F. Denmark
- G. France
- H. Germany
- I. Great Britain
- J. International
- K. Italy
- L. Miscellaneous data
- M. Poland
- N. Russia
- O. South America
- P. United States of America
  1. Army
  2. Navy

LXXXIII. Stereopsis (see depth perception)
LXXXIV. Teaching
LXXXV. Tension, intra-ocular
- A. Altitude (see altitude-anoxia—tension)

LXXXVI. Therapeutics
- A. Air sickness
- B. Color vision
- C. Cornea
- D. Dark adaptation
- E. Decompression
- F. Fatigue
- G. Glaucoma
- H. Injuries
  1. Burns
  2. Foreign bodies
- I. Miscellaneous data
- J. Muscle imbalances
- K. Optic nerve
- L. Psychiatry (see psychiatry)
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- N. Syphilis
- O. Vitamins

LXXXVII. Tobacco
- A. Amblyopia (tobacco-alcohol)
- B. Anoxia
- C. Conjunctiva
- D. Fatigue

E. Miscellaneous information
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G. Retina
H. Vision
I. Visual reaction time

LXXXVIII. Transportation of aviators with head and eye injuries

LXXXIX. Vertigo (ocular)

XC. Visual acuity
A. Acceleration (see acceleration-deceleration—visual acuity)
B. Altitude (see altitude-anoxia—visual acuity)
C. Defects (see also disqualifying defects—visual acuity)
D. Depth perception
E. Examination (see also examination—visual acuity)
F. Eye movements
G. Goggles (see goggles and eye glasses)
H. Illumination
I. Miscellaneous information
J. Signals
K. Tobacco (see tobacco-vision)

XCI. Visual efficiency

XCII. Visual fields
A. Acceleration (see acceleration-deceleration—visual fields)
B. Altitude (see altitude-anoxia—visual fields)
C. Asthenopia
D. Color vision (see color vision—visual fields)
E. Defects
F. Examination (see examination—visual fields)
G. Goggles (see goggles and eye glasses)
H. Miscellaneous information
I. Scotomas

XCIII. Visual reaction time
A. Acceleration (see acceleration-deceleration—visual fields)
B. Accommodation (see accommodation—speed of adjustment)
C. Altitude (see altitude-anoxia—visual reaction time)
D. Examination (see also examination—visual reaction time)
E. Miscellaneous information
F. Standards
G. Tobacco (see tobacco—visual reaction time)

I. Acceleration—Deceleration

A. BLACK-OUT—RED-OUT

1. ANDINA, E.: *Black out as expression of variation in blood pressure during "nose-dives" (Ueber "Schwarzsehen" als Ausdruck von Blutdruckschwankungen bei Sturzflügen)*, Schweiz. med. Wchnschr., 67:753-56, 1937.

The author, with a military flier as pilot, observed and recorded his own experience with reference to seeing black while the plane made the loop, half-loop, and sudden long drop. According to intensity, the visual disturbances may be divided into three phases. The first may be termed "seeing darkly," with the reading matter looking dark but still legible. At first there is no scotoma. In the second phase a black curtain appears in front of the eyes, beginning at the nasal side, with a portion of the field of vision completely eliminated. In the third phase there is completely blackness, total amaurosis. The three phases can hardly be said to follow strictly one after the other because they merge so quickly. Even during the last phase there are no signs of impaired consciousness. The eye seems to be more sensitive after the subject has been flying on his back with head down. In this experimental flight the pilot's reactions were the same as those of the author.

With the aid of another physician, the pressure conditions, etc., of the flight, as affecting the eye, were reproduced by dynamometry, according to Bailliart. The visual disturbances were completely analogous in character to those experienced during the flight. In order to induce the three phases of visual disturbances, the intra-ocular pressure had to be increased by the dynamometer to the following values (reduced to mm. Hg.): "seeing darkly," to 44 mm. Hg.; appearance of the curtain (loss of nasal field of vision), to 51 mm. Hg.; complete amaurosis, to 75 mm. Hg.

Study of the fundus oculi in connection with these findings showed that the pressure conditions responsible for the visual disturbances were localized in the regions of the basal cerebral vessels. It was found that under the action of flight with a force amounting to 5 g., the arterial pressure in the central retinal artery drops from 78 to 21 mm. Hg., and the flier experiences complete amaurosis.

2. ANON.: *Another kind of blackout*, Lancet, 2:1125-26, 1939.

High speed flying often results in transient blindness (blacking out or amaurosis fugax), brought about by sudden change of direction or when power diving.

Forces meeting the pilot in a straight anteroposterior or lateral direction while in flight do not have the same results as when accelerating in a curved path. Here the body may be subjected to a centrifugal force with the head inward, or less frequently outward. A force of more than 5 g. causes diminution, or loss of vision, rapidly followed by loss of consciousness. The changes are due to centrifugal removal of the blood from the brain.

The ability to withstand such stress depends on the power of the cardiovascular system to adjust itself to the forces encountered.

Whether the blindness is due to retinal ischemia or vascular alterations in the visual

pathway has not yet been determined; however, Livingston brought forward evidence against the theory that ischemia results from occlusion of the retinal arteries by positive intra-ocular pressure, since this is unchanged by centrifugal action. He believed that alterations take place in the neighborhood of the external geniculate bodies and the area striata.

3. BÉHAGUE, P. AND MOTHON: *Visual disturbances following accelerations during flight: "Anopsia of aviators" (Troubles de la vision lors des accélérations durant le vol: "L'anopsie des aviateurs").* Rev. neurol., 67:197-201, 1937.

The authors are concerned with "black vision" of aviators, a transient blindness occurring only with rapid variations in speed in either direction. High speed alone is not sufficient to produce the condition; the accelerations, most often negative, must be of the order of 5 to 6 g. at least, as below that nothing happens. The unit of acceleration of a free-falling body being 9.81 meters per second, it is necessary that the speed pass from zero to 49.05 or 58.86 m. per second (or better that it be reduced from this figure per second) to cause "black vision."

The authors quoted statements of six or more pilots, racers, or stunt fliers, who have experienced this condition. Close turns also cause it, and the authors stated that centrifugal force also is an element in the problem. They believe this anopsia of aviators is due to circulatory disturbance and cite the experiments conducted by Broca and Garsaux in centrifuging dogs. In order to produce their death it was necessary to have accelerations of 84 to 97 g., and at autopsy the mesenteric vessels were found distorted and dilated to the size of the little finger while the brain was exsanguinated.

The authors stated that as pilots they are going to continue their studies to determine whether the condition is one of cerebral ischemia or disturbance of the retinal circulation.

4. BLAKEMORE, C.: *Visual complications of high-speed flying.* Tr. Ophth. Soc. Australia, 2:28, 1940.

The visual complications that may occur fall roughly into two groups: (a) sudden transient blindness, or some modification thereof, with or without a train of cerebral symptoms, such as fainting, coma, etc.; (b) retinal and subconjunctival hemorrhages. Of these two groups the former not only is met with more frequently but is the more important. This type of transient blindness is known colloquially as a "black-out." A "black-out" may occur in an individual if, while flying, sufficient corticifugal centrifugal force is created and provided always the exposure to this force is of sufficient duration. The "black-out," however, does not come on immediately; an individual is subjected to the effects of a suitable centrifugal force, but there is a latent period which varies in length in different individuals. This latent period is followed by a period of complete blindness, after which sudden recovery takes place.

The experimental evidence appears to suggest that the "black-out" threshold is reached when a corticifugal centrifugal force of from 4.5 to 5 is applied for from 5 to 15 seconds.

Resistance to "black-out" conditions can be increased by constant flying practice, but the tolerance established is very limited and depends on (a) individual idiosyncrasy, and (b) conditioning of the blood-vascular system to the sudden onset of centrifugal force.

5. DIRINGSHOFEN, H. VON: *Medical Direction for the Air Force. With an appendix on first aid in aviation accidents (Medizinischer Leitfaden für fliegende Besatzungen. Mit einem Anhang Erst Hilfe bei Flugunfaellen)*. Dresden and Leipzig, Theodor Steinkopff, 1939, 204 pp.

This volume is written for the aviator so that he may have a knowledge of aviation medicine and thus may guard his health and increase his flight efficiency. Reports of pilots concerning the visual disturbances occurring at great speed and from centrifugal force are inaccurate because loss of consciousness may occur simultaneously. Normal disturbances (lasting only a short while) are: "a gray veil" at first; then borders of objects look blurred as if seen through fog, and at last complete loss of vision, called "blacking out." Consciousness may remain undisturbed, or unconsciousness may occur, becoming normal again, as soon as conditions return to normal. Investigations showed that disturbances of the circulation of the blood in the retina and brain are responsible. As the weight of blood increases according to centrifugal force, differences in blood pressure and lack of oxygen occur. Decreasing blood pressure affects the eye more than the brain because of the eye tension which in the case of low blood pressure compresses the fine vessels. A diagram is used to show that the higher an organ is located in the body, the lower the blood pressure in its arteries.

Visual disturbances occur if the blood pressure of the brain arteries is less than 50 cm. W. According to reports, the upper limit of centrifugal force tolerated without visual disturbances is approximately between five- to six-fold of gravity.

In looping, conditions are different. The blood pressure is too high, and subconjunctival and even retinal hemorrhages occur.

A survey concerning the importance of the eye and its protection is given. Sight should be perfect and corrected by glasses if necessary.

Tinted glasses absorb the bright light and make contrasts between red and green more distinct.

A pilot should not be color blind because of colored signals. Experience shows that protanomalous and deuteranomalous individuals confuse red, green and white more than usual under special conditions of light as well as when tired or when there is a lack of oxygen.

Partly tinted glasses are recommended.

Hallucinations occur in blind flight and the pilot must rely on his technical instruments only.

There are chapters on the following: origin and treatment of air sickness, sport and training, fitness, and health and its preservation.

6. LIVINGSTON, P. C.: *The problem of "black-out" in aviation (amaurosis fugax)*. Brit. J. Surg., 26:749-56, 1939.

The first impression derived from the terrific impact of the aircraft against the resistance of the air at the point of turn is one of intense bodily strain producing what might be described as a "concertina sensation."

Loops out of a dive may produce only discomfort with a suspicion of visual uncertainty at 200 m.p.h. There may be dimness of perception of the ground, and a sensation may be

caused as though the accommodation had been overtaxed by bringing print too near the eyes.

A favored view of causation of "black-out" is one based on the compressing influence of intra-ocular pressure upon the central retinal artery and its branches. The vessels are believed to collapse because the systolic drive in the ophthalmic artery is seriously impaired, and the intra-ocular tension of 20 mm. of mercury provides an impassable resistance. Thus, retinal nutrition is brought to a standstill. (The author does not agree fully with this view.)

The retina is peculiar in so much as it is more dependent upon oxygen for the maintenance of its vitality and function than any other tissue within the body. It was found that the oxygen demand represents a four-fold value over that of other structures. Were the retina deprived completely of its blood supply and were the vessels actually compressed to extinction, there would be a definite risk of permanent damage to the visual receptor mechanism. This has never been recorded. The process may not be one of complete vascular denudation, but rather retardation of the existing flow.

Free use of oxygen has little effect on the onset of "black-out," while CO_2 seems to be beneficial.

7. MARSHALL, G. S.: *Physiological problems of human flight*. Lancet, 1:270-71, 1940.

The physiological problems of flight are divided into those of position and those of movement. "Blacking out" is the most common symptom of excessive acceleration. The effect can be alleviated by bending the body forward and tightening the muscles. The pilot faints when the pressure is continued for a long time. The movement of the body fluids into the splanchnic area cause the symptoms. Temporary blindness is probably caused by a failure in the arterial pressure in the neighborhood of the eye to a point at which the blood can no longer overcome the intra-ocular pressure of from 20 to 25 mm.

8. PHILLIPS, R. B. AND SHEARD, C.: *Amaurosis Fugax: effect of centrifugal force in flying*. Proc. Staff Meet., Mayo Clin., 14:612-18, 1939.

The transitory or fleeting blindness in amaurosis fugax lasts from 1 to 20 seconds, being occasioned by sudden change of direction at high speed. Transverse accelerations up to 16 g. cause no untoward effects on experimental animals and, up to 10 g. do not produce disturbances of vision. Armstrong and Heim found that, flying in the vertical (positive) direction, in man, vision was lost after 5 g. was attained; coma or fainting developed between 6 g. and 9 g. Tears were thrown off in a stream at —4 g., and at —4 g. and —4.5 g. there were hemorrhages into the conjunctival membranes and a feeling that the eyes were protruding from their sockets. The return of vision occurs suddenly and dramatically.

Generally the loss of sight is attributed to the compression exerted by the intra-ocular pressure on the central retinal artery and its branches, but various facts point to the conclusion that the local conditions in the eyes form only part of a chain of factors affecting the lower visual centers and the corticovisual areas concerned with conscious mental processes.

The retinal vessels are believed to collapse because the systolic pressure in the ophthalmic artery is markedly reduced under centrifugal action, and the intra-ocular pressure of 20 mm. of mercury offers a resistance which cannot be overcome.

The process may be due to retardation of the existing flow of blood. The attack may not be concentrated on the retina alone, but it may be spread throughout the visual pathway.

9. Rook, A. F. and Dawson, D. J.: *Hypotension and flying.* Lancet, 2:1503-10, 1938.

In flying tests distant vision was found to be impaired in a number of cases, but occasionally distant vision remained clear, whereas close objects appeared blurred.

The most common symptom in flying is that known as "blacking out." This varies from a dimming or graying of the whole field of vision to complete blindness, often with no effect on any of the other systems of the body. In one case a central scotoma was experienced, and one subject described the loss of only the peripheral field. In most cases the loss of vision was gradual and even throughout the field. One aviator described the effect as similar to the drawing of a blind over the field of vision or to the closing of a diaphragm of a camera.

10. Whittingham, H. E.: *Preventive medicine in relation to aviation.* J. Roy. Army M. Corps, 73:278-81, 1939.

The author described "blacking out" resulting when the pilot makes sharp turns at high speed and centrifugal forces greater than 4 g. are developed. The first symptom is the feeling of being forcibly pressed into the seat of the plane. Then increasing dimness of the visual field, and sudden blindness ensue. The "blacking out" lasts from 2 to 5 seconds depending on the force and the duration of the "g." The phenomenon is considered due to the centrifugally induced splanchnic pooling of the blood. There is a reduction of blood pressure in the head, especially in the central retinal artery, with a temporary interference with the retinal circulation.

The preventive measures suggested are: (1) keeping flying personnel fit, (2) use of belts to support the abdominal wall, and (3) the adoption of a crouching posture by the aviator.

See also: No. *16*, von Diringshofen, 1940; No. *153*, Sédan, 1938; No. *309*, Fulton, 1940; No. *408*, Longstreth, 1929; No. *723*, Ryan, 1940; No. *1017*, Whittingham, 1940.

B. EFFECTS (MISCELLANEOUS) OF ACCELERATION—DECELERATION

11. Archangielskiy, A.: *Pilots and acceleration.* Vo.-sanit. Dyelo, 1939.

12. Bergeret, P. M.: *Physiologic effects of speed and accelerations in flying [Effets physiologiques de la vitesse et des accélérations en avion].* Rev. Serv. de San. mil., 107:301-24, 1937.

13. Bergeret, P. and Gougerot, L.: *Vestibular effects of acceleration on the aviator [Effets vestibulaires des accélérations chez l'aviateur].* Travail hum., 7:1-26, 1939.

The effects discussed include the circulation of the eye which may result in distortion of vision and lead to accident.

14. Bührlen, L.: *Studies on the importance of direction in the action of force on the body during flying (Versuche über die Bedeutung der Richtung bei Einwirken von Fliehkräften auf den menschlichen Körper).* Luftfahrtmed., 1:307-25, 1937.

Large increases in speed cause visual disturbances. In the flat position (flying direction chest-back) visual disturbances occurred, but the brain and consciousness were not affected. Experiments were made in the sitting and lying positions.

15. Diringshofen, H. von: *Increased danger of circulatory collapse due to simultaneous action of altitude and acceleration in airplanes (Untersuchungen im Flugzeug über die erhöhte Kollapsgefahr durch gleichzeitige Einwirkung von Höhe und Beschleunigung).* Verhandl. d. deutsch. Gesellsch. f. Kreislaufforsch., 13:92-97, 1940.

16. Diringshofen, H. von: *Medical guide for flying personnel,* translated by V. E. Henderson, University of Toronto Press, 1940.

This monograph considers high flights, the effect of acceleration and of centrifugal force, the importance of the sense organs for the flier, sensory illusions in blind flying, the effects of noise and vibration and the cause of air sickness, sport for flying personnel, hygiene, the important points in the medical examination of flying personnel, and the flier and his medical officer.

Persons sitting in an airplane may suffer the following disturbances:

(1) Those occurring during the normal increase in centrifugal force which is of short duration.

(a) The visual disturbances which occur in one or two seconds after the force is applied and quickly disappear as the centrifugal force diminishes. These are the well known "gray veil" or "mist before the eyes" which, if the force is greater, becomes the "blackout," the total loss of vision with consciousness retained.

(b) The same disturbance together with temporary loss of consciousness.

(2) The deep, long-lasting loss of consciousness due to the centrifugal forces overstraining the circulation and leading to a circulatory collapse (collapse due to excessive strain).

(3) A very transitory disturbance of vision with a slight clouding of consciousness after a short zoom upward.

The disturbance of vision and consciousness most frequently observed occur only one and one-half to two seconds after the beginning of the centrifugal force and end almost with its cessation. Dependent on the magnitude of the force the disturbances are more or less marked.

A very slight disturbance consists in a "gray veil." The outlines of the objects under observation, instruments, etc., are no longer sharp. They appear as though they were seen through a mist, which becomes thicker as the force increases and thins as it decreases. With increasing centrifugal force the mistiness passes into blindness, which is described as the eyes becoming black or "blacking out." Consciousness is quite clear and thinking undisturbed.

There is no longer any doubt that disturbances of blood flow in the retina of the eye and in the brain are the cause of the disturbances.

Every flier who can improve his vision by the use of glasses should make use of them and should become accustomed to using them when flying.

Goggles protect against the wind. They must be a good fit and must particularly close off the inner angles of the eyes so that the air current may not damage the outer surface of the eyeball and lead to a chronic inflammation of the surface.

Anyone who is sensitive to bright light may wear colored glasses in his flying goggles.

17. FISCHER, U.: *The circulation under the influence of acceleration. X-ray pictures of conditions in apes (Der Kreislauf unter Beschleunigung. Roentgenaufnahmen beim Affen).* Luftfahrtmed., 2:1-13, 1937.

In 10 apes (Macacus rhesus) acceleration experiments on the centrifuge were performed after the injection of thorotrast. Simultaneous x-ray pictures were taken with the aid of an accompanying x-ray arrangement. There are no eye findings in this article.

The power of resistance of the animals corresponded to their physical condition.

18. GAUER, O. AND RUFF, S.: *The limits of tolerance for flying stress in the transverse direction (Die Ertraglichkeitsgrenzen fur Fliehkrafte in Richtung Rucken-Brust).* Luftfahrtmed., 3:225-50, 1939. Abstract: J. Aviation Med., 10:156, 1939.

Human subjects can tolerate for longer periods (more than 30 seconds) transverse flying stress from back to chest, corresponding to centrifugal acceleration of 8-10 g. The subjective symptoms are so slight that it appears likely that this limit might be considerably exceeded, especially for short periods.

In prolonged centrifugal tests above 8 g., petechial hemorrhages appear in the ocular conjunctiva which disappear after two days.

The degree of tolerable transverse stress in the back-chest direction depends largely upon the condition of the substrate upon which the body rests and upon the construction of an apparatus giving reliable support to the head at onset of acceleration and prevents injury by knocking against other structures.

19. KOENEN, R. AND RANKE, O. F.: *The circulation under the influence of acceleration. Intravascular determination of the blood pressure in the dog (Der Kreislauf unter Beschleunigung. Blutige Blutdruckmessung am Hund).* Luftfahrtmed., 2:14-26, 1937.

In 6 anesthetized dogs the carotid blood pressure, under the influence of flight force exerted in longitudinal axia of the animal, was determined.

In the anesthetized dog the blood pressure drops as soon as acceleration begins (flight force in the gluteal region) and rises under lower flight force within 18 to 20 seconds, in accordance with the reflex time of the carotid sinus. In flight force with acceleration beyond 4 g., the carotid blood pressure remains constantly below zero.

The pulse frequency drops in the dog as soon as the action of flight force sets in, in spite of good functioning of the carotid sinus. This is due to an unexplained vagus stimulus. The drop in blood pressure under the action of flight force is due to a reduction in the quality of circulating blood, caused by displacement of the blood into the parts of the body which are hydrostatically in lower position. Upon cessation of acceleration, the blood returns to the heart within a few seconds.

There is no discussion of conditions in the eye.

20. PEDRAZZINI, F.: *Concerning the cerebrospinal circulation especially in regard to the effects that the centrifugal force exercises on this system and on the general circulation of aviators (De la circulation cérébro-spinale particulièrement par rapport aux effets que le force centrifuge exerce sur ce système et sur la circulation générale chez les aviateurs).* Presse méd., 2:1164-67, 1938.

The author quoted Heinz von Diringshofen as having noticed during his investigations: a sensation of darkness, narrowing of the visual field like that of a beginning narcosis, loss of consciousness, variations of arterial pressure and pulse frequency, with sanguinary engorgement of the lower limbs.

The author's own work was on the effect of centrifugal force on the circulatory system, and there is no specific mention of the effect on the eye.

21. SCHUBERT, G.: *The physiological effects of the coriolis forces during rolling of the plane (Über die physiologischen Auswirkungen der Corioliskräfte bei Trudelbewegung des Flugzeuges).* Acta otolaryng., 16:39-47, 1931.

Coriolis forces are forces due to a coriolis acceleration. Accelerations perpendicular to the track of the relative movement occur in all relative movements in which the system rotates. There are no prescribed movements for the pilot because, depending on the movements of the plane, a certain movement of the head may hinder or help, for it may produce delusory perceptions which would prompt the pilot to steer the plane the wrong way. It is desirable to keep still and to try to keep looking at the horizon or the earth's surface.

22. WÜRDEMANN, H. V.: *Problems arising from the effect of high speed on living tissues.* J. Aviation Med., 6:27-29, 1935.

It has been estimated that the extreme speed which can be reached by airplanes is 660 miles an hour. This is based upon the fact that air, moving at this rate, disintegrates the association of its molecules. The high droning sound of an airplane is said to be due to this effect upon the air by the ends of the propellers traveling at the rate of 1,250 to 1,500 miles an hour, which cause the molecules to bump against each other.

Living tissue cannot stand the strain of extreme speed, and it is doubted that turns can be made with safety at a speed of 660 miles an hour. Many pilots have made dives at the rate of 400 miles an hour; however, in rapid turns, descents, etc., some pilots have been incapacitated from brain and abdominal hemorrhages and have lost sight through hemorrhages into the retina and cornea.

See also: No. 192, Tschermak-Seysenegg, 1933-34; No. 333, Clements, 1923; No. 726, Strughold, 1938.

C. RETINAL BLOOD PRESSURE

23. DIRINGSHOFEN, H. VON: *Effects of linear acceleration and centrifugal forces on man (Die Wirkung von gradlinigen Beschleunigungen und von Zentrifugalkräften auf den Menschen).* Ztschr. f. Biol., 95:1-26, 1934.

Von Diringshofen showed that hemostatic forces cause visual disturbances in pilots during quick turns (centrifugal forces). A disturbance of circulation in the brain and retina

due to hydrostatic blood pressure and a lowering of pressure in the brain arteries as compared to the pressure in the heart region is the basis for this reaction. He has devised a method of computing the hydrostatic changes in blood pressure in each bodily region as dependent on the acceleration acting on the body. Thus the acceleration at which each individual will have disturbances of vision or of consciousness can be plotted.

After explaining the practical significance of investigations on the physiologic effect of linear accelerations and centrifugal force on man, the author covered these accelerations in an introduction on physics, with particular attention to the forces which act upon the crew of an airplane in flight. He presented a few handy formulas for calculating the acceleration forces and gave a brief review of the most important flight figures and flight conditions, in which centrifugal forces have a noteworthy effect upon the organism of the flier and especially upon his circulatory system. These effects were studied by the author theoretically and experimentally by performing curves and steep spirals in flight.

In this first communication he presented the hemostatics of the circulation of the blood under the influence of acceleration. He placed special emphasis upon the importance of hemostatic consideration as a basis for the investigation of hemodynamics of the circulation under the influence of accelerations. The author showed that the visual disturbances, observed by the flier as a result of centrifugal forces at the time of rapid changes in flight direction, may be explained solely on the basis of hemostatic considerations. According to the author's opinion these visual disturbances are due to a circulatory disturbance (hyphemia) in the brain or in the retina of the eye as a result of hydrostatic reduction in blood pressure in the cerebral arteries as compared with the blood pressure at the level of the heart.

By means of a schematic arrangement the hydrostatically changed pressure for the blood vessels of every part of the body, as dependent upon the degree of acceleration, may be read or computed. The hemostatically induced changes in blood pressure in the veins and arteries are explained as to their practical effects in normal flying and up-side-down flying.

In the next communication (II) the author will give the results of simultaneous investigation of blood pressure, frequency of heart beat, and respiration under the influence of high accelerations in curve and steep spiral flights. He will explain his method of electrocardiography, continuous recording of blood pressure and pneumotachography in man during motor flight.

In these investigations the "indivdual basic curves" of blood changes at heart level, dependent upon the degree of acceleration, were obtained for various periods of acceleration. These curves represent a certain "norm" and may give information concerning the ability of the individual to bear high accelerations, by way of showing at what degree of acceleration disturbance of vision and consciousness may be expected.

24. Uyemura, M. and Ishida, A.: *Hydromechanic studies on the blood pressure of the retinal blood vessels, Nippon Gankagakkai Zassi.* (J. Jap. Ophth. Assn. [Acta Soc. Ophth. Japan]), 44:1114-39, 1940. Abstract: J. Aviation Med., 12:266, 1941.

Four healthy males were measured for the capillary blood pressure of the macula lutea in different body positions by means of Uyemura and Sugnanuma's ophthalmodynamometer. The retinal blood pressure in a horizontal position was higher than in a vertical

position and increased by 3.9, 10.7, 13.7, and 19.6 mm. Hg., respectively, when the body was tilted head downward at a certain speed to angles of 15, 30, 45, and 60 degrees.

The brachial blood pressure, which was measured simultaneously, did not change at any position. The rise of the retinal blood pressure was due to the stagnation of blood in the retinal capillaries brought about by centrifugal force. With repeated experiments the rise became less and less noticeable as a result of the adaptation of the subject. When the tilting was done at a slow and constant rate, no rise in the retinal blood pressure was observed. By contrast, when the tilting was done suddenly and rapidly, the retinal blood pressure rose twice as high as normal. Hence the blood pressure in the retinal capillaries is subject to hydrodynamic principles and is changed by centrifugal force when the body is in motion, unless compensated by physiologic adaptation.

See also: No. 1, Andina, 1937; No. 2, Anon., 1939; No. 3, Béhague & Mothon, 1937; No. 4, Blakemore, 1940; No. 6, Livingston, 1939; No. 8, Phillips and Sheard, 1939; No. 153, Sédan, 1939.

D. VISUAL ACUITY

25. DIRINGSHOFEN, H. VON: *Studies in a plane of the effect of body position on the ability to endure great accelerations (Untersuchungen in Motorflude über den Einfluss der Körperhaltung für das Ertragen hoher Beschleunigungen)*, Luftfahrtmed., 1:226-40, 1936.

In an upright position of the torso, visual disturbances appear at a velocity of 450 km. per hour with a centrifugal force over 4.5 g. (flying spirals straight down). Disturbances of vision occur at a centrifugal force above 5 g. In a crouching position no disturbances occur up to a centrifugal force of 6 g., even if the force lasts for 10 to 15 seconds.

26. RUFF, S.: *On the behavior of blood pressure and pulse rate under the influence of flight forces; also on experiments with increasing tolerance for acceleration (Ueber das Verhalten von Blutdruck und Pulsfrequenz unter dem Einfluss von Fliebkräften und über Versuche zur Steigerung der Beschleunigungserträglichkeit)*, Luftfahrtmed., 2:259-80, 1937-38.

The author describes a method by which systolic and diastolic blood pressure may be registered about every 15 seconds, even under the influence of high acceleration.

With this method experiments were performed on man, using the centrifuge, in connection with which the blood pressure and pulse were registered. These experiments showed that accelerations up to 3 g., even for considerable periods of time, were tolerated without disturbances and with no essential changes in blood pressure. The pulse, however, rose rapidly from the beginning in this as well as in all other experiments. In the presence of accelerations over 3 g. disturbances to vision and consciousness occurred, accompanied by considerable change in blood pressure. The changes in blood pressure and pulse readings threw light upon the failure of the organism under the influence of acceleration and also upon the scope of self-adjustment of the circulation.

In experiments with accelerations up to 5 g. for brief periods, there were only slight changes in pulse rate. Reduction of systolic blood pressure and of blood pressure amplitude occurred only after 4 g. had been reached. There were no subjective disturbances in this group of tests. Further experiments had to do with determining the effectiveness of a number of measures for increasing the acceleration tolerance. It was found that during

digestion the tolerance was greatly increased and that addition of CO_2 to the atmosphere (within the plane) was also helpful. The use of abdominal girdles was not quite so effective. Another measure which increases tolerance to acceleration is lying down. In this position high speed does not affect the organism so much as in the sitting position.

27. SHADEE, J.: *Effects of speed on visual acuity in air pilots (Les modifications de l'acuité visuelle chez les pilotes après les vols de chasse).* Rev. Pol. Med. Aeronaut., No. 1, 1936. Abstract: J. Aviation Med., 7:217, 1936.

The author divides his material into two age groups. In the older group (29-36 years) after prolonged flights of 45 minutes to one and one-half hours there occurred a diminution of visual acuity persisting for one to two hours. No such diminution was noted after short flights. In the younger group visual acuity was, on the contrary, increased following flight, regardless of duration. The changes in visual acuity were insignificant, averaging only about 0.05. The author believes them due to changes in the circulation of the blood occurring in rapid flight at high altitude owing to accelerated changes in direction of flight. These changes are better compensated and disappear sooner in the younger pilots.

See also: No. 9, Rook & Dawson, 1938; No. 715, Ballantyne, 1940.

E. VISUAL FIELDS

28. BREUER, J. AND KREIDL, A.: *On the apparent rotation of the field of vision under the action of centrifugal force (Ueber die scheinbare Drehung des Gesichtsfeldes während der Einwirkung einer Centrifugalkraft).* Pflüger's Arch. f. d. ges. Physiol., 70:494-510, 1898.

The centrifugal force considered, among others, is that generated by a moving train, and the seeming rotation or tilting of the field of vision is evident in the seemingly slanted position of objects, such as telephone poles, when seen through the train windows.

The authors made a careful study of these phenomena and corroborated the findings of Purkynie and Mach, who stated that the subject feels the direction of the mass acceleration. If this direction is changed by a horizontal acceleration acting upon the body from the side, rotation of the eyes takes place. This rotation persists throughout the action of this second force and amounts to half (0.6) of the deflection angle. Rotation of the field of vision and slanted position of vertical lines, which are noted under such circumstances are due, therefore, to actual unconscious rotation of the eyes.

See also: No. 1, Andina, 1937.

F. VISUAL REACTION TIME

29. BURMEISTER, H.: *Investigations on changes of optic reaction time under the influence of great flight forces (Untersuchungen über Änderungen der optischen Reaktionszeit des Menschen beim Einwirken hoher Flughräfte).* Luftfahrtmed., 3:277-84, 1938-39.

In 8 experimental subjects the author performed 17 experiments on changes of optic reaction time under the influence of flight forces as created on the centrifuge.

When the flight forces acted in frontodorsal direction, at 4 g., 5 experimental subjects

showed slight increase of the reaction time. At 8 g. the reaction time was decidedly lengthened.

When the flight forces acted in head-seat direction, an increase in reaction time was noted at 3 g. At 4.5 g. these changes were even more distinct, and in some instances the accompanying visual disturbances induced omission of reactions.

II. Accidents

A. CAUSES (MISCELLANEOUS)

30. BENJAMIN, J. D.: *An analysis of aviation crashes.* U. S. Nav. M. Bull., 24:72-75, 1926.

The cause of any crash should come under one of the following four headings: (1) faulty piloting; (2) mechanical; (3) unavoidable (collisions, weather conditions) and (4) unknown. In this paper no reference to the influence of eye defects on aviation accidents is made.

31. BENJAMIN, J. D.: *Crashes at Pensacola, 1927.* U. S. Nav. M. Bull., 26:63-69, (Jan.) 1928.

Statistics show that aviation is steadily becoming safer. The physical examinations have become stricter or the system of checking the students is more severe because the percentage of those finishing instruction is steadily decreasing. Within two years (since 1925) flying has become as much as 50 per cent safer. According to statistics, February always was the safest month; however, it must also be taken into account that it is the shortest month with two holidays.

In Pensacola, in 15 out of 26 crashes, inexperience was the cause; 5 people were killed and 7 injured.

There is no reference to the eye in this paper.

32. CERES, F.: *Flight hazards in naval aviation.* Mil. Surgeon, 71:225-30, 1932.

The causes of accidents for the years 1929-1930 were given in the report of the Assistant Secretary of the Navy for Aeronautics on March 16, 1931. The human factor was responsible in 54.59 per cent of the accidents. The immediate causes of pilot error included error of judgment—33 per cent.

There were 14 fatal accidents; in 33.93 per cent error in pilot judgment was the immediate cause.

33. GILCHRIST, N. S.: *An analysis of causes of breakdown in flying.* Brit. M. J., No. 2: 401-403, 1918.

Of 100 consecutive cases rejected by the Special Medical Board as "permanently unfit" for flying, visual difficulties, mainly landing, were present in 4 per cent.

See also: No. *54*, Ferree & Rand, 1937; No. *180*, Anon., 1937; No. *356*, Berens & Uren, 1920; No. *382*, Benjamin, 1929; No. *383*, Cooper, 1931; No. *402*, Forster, 1920; No. *430*, Padden, 1930; No. *638*, Ferree & Rand, 1938; No. *665*, Anon., 1924; No. *827*, Ruff, 1937; No. *1007*, Armstrong, 1937; No. *1119*, Sinclair, 1931.

B. DEPTH PERCEPTION

34. ANON.: *Congress of Ophthalmology. Italian Letter.* J. A. M. A., 91:1304-1305, 1928.

Pastor presented a study of 50 air pilots which showed depth perception to be due mainly to binocular parallax and to the size of retinal image. Almost all flying accidents are due to faulty perception of distance.

35. VAN WULFTEN PALTHE, P. M.: *Aviation accidents and their causes (Vliegongevallen en hun Oorzaken).* Nederl. tijdschr. v. geneesk., 65:3264, 1921.

Of 27 accidents occurring among 35 aviators, the largest percentage was caused by faulty judgment of distance. Aviators making bad landings were found to be defective in estimating distance by binocular parallax alone. Therefore, the author believed that this should be kept in mind in the selection of aviators, and that this function should be trained. He also recommended that physicians should fly themselves so that they may be better acquainted with the problems of aviation medicine.

See also: No. 323, Pastore, 1927; No. 334, Colajanni, 1939; No. 512, Howard, 1920.

C. HETEROPHORIA

36. SPALTH, E. B. (Abstractor): *Papers presented at the meeting of the British Medical Association in which points of importance to the air service medical officer were discussed.* Mil. Surgeon, 53:503-506, 1923.

The author has abstracted papers presented at a meeting of the British Medical Association held on July 27, 1923. Intra-ocular as well as extra-ocular normalcy was stressed by Munro. Heterophoria was observed in almost all chronic bad landers. Clements observed that from 70 to 80 per cent of accidents occurring at training camps were due to bad landings. The chronic offenders were found to come from the same stations and trades in civil life which had given the greatest number of industrial accidents. Clements believed the accidents were caused mainly by heterophoria made manifest by fatigue. To correct this condition he recommended amblyoscopic exercises, prisms and stereoscopic exercises. He used the pencil convergence test and cover test at 20 inches to check on the examination and treatment. The cover test indicates the recovery power because the speed with which the deviating eye returns to fixation is observed. He used red and green lenses. The Bishop Harman diaphragm test was used for muscle testing.

The following table shows the relationship of heterophoria to flying in 149 chronic offenders and 124 good pilots:

	Bad Landers	Good Fliers
No heterophoria	1 per cent	99 per cent
Borderline condition	4	1
1° heterophoria	13	0
2° heterophoria	18	0
3° heterophoria	56	0
4° heterophoria	8	0

See also: No. 37, Anon., 1921; No. 39, Kice, 1931; No. 40, Truby, 1922; No. 43, Clements, 1925-26; No. 371, Cooper, 1930; No. 508, Dudley, 1919.

D. STATISTICS

37. ANON.: *Statistical report on airplane crashes in the air service during 1920 (Office of the Chief Surgeon, Air Service).* Air Serv. Inform. Circ. 4, No. 340:4-6, 1921.

An analysis of 312 crashes was made. The statistical table on physical defects of pilots who crashed as determined by last prior physical examination is as follows:

	Crashes		Crashes
No physical defects	284	Ear drums retracted	1
One eye 20/20 to 20/30	3	Unstable nervous system	2
Both eyes 20/20 to 20/30	1	Elbow deformity	1
One or both eyes less than 20/30	1	Color blindness	1
Defective depth perception	1	Nystagmus	1
Esophoria	1	Otitis media	1
Exophoria	1	Nasal obstruction	2
Hyperphoria	5	Hypertrophied tonsils	2
Hyperphoria and esophoria	1	Defective teeth	1
Low prism divergence with esophoria	1	Perforated ear drum	1
		Total	312

The flight surgeon reported the cause of crash as misjudged landing in 19 crashes, and as pancaking in 2 crashes.

38. ANON.: *Effects of physical deficiencies upon flying activities of licensed pilots and holders of student permits.* Air Commerce Bull., 3:113-17, 1931.

The accident rate among physically normal pilots is compared with that among pilots with various types of defects. The per cent of licensed pilots who are physically defective is 11.9 and 88.1 per cent are normal. The defective group was responsible for 17 per cent of all accidents, showing that physical defects introduce a distinct hazard into flying.

Of 29,865 cases the rates for eye defects are given: (a) Minor defects. Percentage of progress in one year or less with: vision (20/20 to 20/40)—17 per cent; defective color vision—14 per cent (ability to make gross color distinctions only). Vision and color vision have a retarding influence approximately equal to the average influence of all the minor defects. (b) Major eye defects. Percentage of progress in one year or less with: vision (20/40 to 20/50)—21.5 per cent; ocular muscle imbalance—4.1 per cent.

39. KICE, L. H.: *Airplane Crashes—Then and Now.* J. Aviation Med., 2:107-19, (June) 1931.

Checking on physical defects among the pilots involved in 91 crashes, the author reported the following: defective ocular muscle balance, 3; defective depth perception, 1; defective visual acuity, 1; defective ocular muscle balance and hearing, 2; and defective visual accommodation, 1.

Kice concluded, from the statistics drawn in 21 crashes accumulated by 12 pilots with defects during a four year period, 12 crashes were registered against 6 different pilots who had defective vision, and in practically every case they "cracked up," due to defective motor or adverse weather conditions, while making a forced landing on a strange field.

This strengthens the theory that where a pilot needs perfect muscle balance and visual acuity the most is when he is attempting to land on a strange field. He is usually fatigued and is landing under bad weather conditions, when ocular muscle weakness and defective visual acuity are most severely tried. All 6 of these pilots, at some time or other during this four year period, had carried waivers for the physical defects in question, that is, defective visual acuity or defective ocular muscle balance.

According to statistics, 100 defective fliers averaged 160 crashes and 100 non-defective aviators averaged 130 crashes over the same period of time.

One case is described in which an experienced flier, considered to be one of the better pilots, suddenly developed phobia for night landings during maneuvers with antiaircraft searchlights, whereupon he had to discontinue night flying for good.

The factors of fatigue, mental stress, too much or too little flying are as important now as they were during the period when this report was compiled.

The necessity of careful physical selection, careful supervision of pilots by flight surgeons, and careful maintenance of planes by thoroughly trained pilots also remains the same.

40. TRUBY, A. E.: *Statistical report on airplane crashes in the air service during the period January 1, 1921 to December 31, 1921.* Air Serv. Inform. Circ., 4, No. 340:7-9, 1922.

An analysis of 330 crashes was made. This report includes statistical tables on the following: crashes by station; number of crashes in each month; crashes according to rank of pilot; crashes according to rating of pilot; age of pilot; crashes according to class of training which pilot had received; hours dual instruction of pilot prior to crash; hours solo flying by pilot prior to crash; hours flown by pilot during month preceding crash; month in which pilot was last physically examined prior to crash; number of previous crashes by pilot; physical defects of pilots who crashed as determined by last prior physical examination; previous flights made by pilot on date of crash; duration of flight before crash; nature of work being performed at time of crash; nature of pilot's duties at field; result of crash for pilot, rank of pilots killed; cockpit of pilot; result of crash for passenger, rank of passenger killed; type of plane; damage to plane; day of week; time of day; weather, terrain; stage of flight at which accident occurred; altitude at which trouble began; distance of site of crash from field; cause of crash as reported by flight surgeon; cause of crash as determined by crash board; arrival of first aid; ambulance.

The table on physical defects of pilots who crashed as determined by last prior physical examination is as follows:

	Pilots
No physical defects	306
One eye between 20/20 and 20/30	1
Both eyes 20/20 to 20/30	2
Hyperphoria	4
Exophoria	1
Angle of convergence	1
Hearing—one ear defective	6
Hearing—both ears defective	1
Unstable nervous system	1
Flat foot	1
Varicocele	1
Overweight	1
Cardiac murmurs	1
Cardiac arrhythmia	1
Hypertrophied tonsils	1
Hypertrophied turbinate	1
Total	330

E. VISUAL ACUITY

41. Anderson, H. G.: *Aeroplane accidents—their medical aspect and their various causes*, Scient. Am. (Suppl.), 87:30-31, 1919.

An analysis of accidents in two schools revealed that poor vision and delayed reaction time may account for many accidents. The causes of the accidents analyzed are listed as follows: defect in airplane, error in judgment in flying, "loss of head," brain fatigue or lethargy, physical illness and unavoidable causes. Of 58 crashes occurring in 9,000 flights with injuries to 16 airmen, error of judgment accounted for 42 crashes.

42. Clements, E. C.: *Ophthalmology in relation to Navy, Army, and Air Force (discussion)*, Brit. M. J., Oct. 13, 1923, pp. 654-56.

Clements described the routine tests used in estimating the heterophorias of aviators. Eighty per cent of crashes occur in making landings and are due to faulty judgment of speed and distance, wherein heterophoria constitutes an important factor. The author recommended the use of eye training as a practical aid to greater proficiency.

43. Clements, E. C.: *Visual problems in regard to flying and industrial fatigue from a service standpoint*, Proc. Roy. Soc. Med., Sect. on War., 9:15-23, 1925-1926.

Pilots who had difficulty in flying, especially in landing, were examined for possible defects in vision. Although all exhibited more or less muscular imbalance or weak binocular vision, or both, the majority had normal vision and refraction.

In certain training schools, unsuccessful landings caused from 75 to 80 per cent of the flying accidents. Difficulties in learning to form judgments in flying are largely the result of altered visual conditions, i.e., loss of accustomed orientation to the ground; foreshortening of shadows of objects; the greatly increased speed; strain on the airman's eyes due to centrifugal force, which, if continued, may under certain conditions render him incapable of correct judgment and even mentally irresponsible.

Clements believed that the symptoms of nervous instability may be induced by heterophoria through the unconscious nervous strain and fatigue brought about by the visual condition in the endeavor to maintain correct cerebral appreciation. However, the ocular muscle imbalance may suggest the presence of a general condition of nervous instability.

44. Jones, G. I.: *The medical division, Army Air Corps*, Mil. Surgeon, 71:422-27, 1932.

Eleven pilots with physical defects crashed. Of these 6 had visual acuity defects and 3 had defective accommodation. The 2 remaining had limitations of joints.

45. Rippon, T. S.: *The personal factor in flying accidents*, U. S. Nav. M. Bull., 26: 149-51, 1928.

Pilots classified as "crashers" include erratic landers with defective vision and those whose vision is defective at night. Certain tests aid in obtaining a thorough knowledge of the individual. The domino test consists of playing a game of dominoes single handed as quickly as possible. A second test of visual apprehension consists of presenting 10

small objects for 6 seconds, after which the subject must enumerate them. A third test is an estimation of reflex time.

See also: No. 13, Bergeret & Gougerot, 1939; No. 179, Wilmer, 1918; No. 371, Cooper, 1930; No. 1079, Brailey, 1919.

F. VISUAL REACTION TIME

46. ANDERSON, H. G.: *The medical aspects of airplane accidents*, Brit. M. J., 1:73-76, 1918.

Faulty reaction time has been the cause of certain airplane accidents. Normal reaction time is 0.19 or 0.20 seconds. It may be delayed by fatigue, drugs and excesses.

47. SELZ, O.: *On the part played by the individual qualities of pilots or observers in airplane accidents (Ueber den Anteil der individuellen Eigenschaften der Flugzeugführer und Beobachter an Fliegerunfällen)*, Ztschr. f. ang. Psychol., 15:254-96, 1919.

Among the qualities to be checked and tested in a pilot are: the speed with which one is able to isolate (or abstract) one object out of a complex (such as a good landing terrain); speed of reaction, capacity to estimate distance; and correct estimation of the result of a movement.

The part of flying which requires most skill is landing.

III. Accommodation

A. AGE

48. DUANE, A.: *Normal values of the accommodation at all ages*, J. A. M. A., 59: 1010-13, 1912.

Several years ago the author presented a report giving measurements of accommodation in 600 subjects of all ages and deducing therefrom a revision of the standards established by Donders for the progress of presbyopia. In making the tests each subject was first rendered fully emmetropic by glasses, and his accommodation was then carefully and repeatedly measured.

Through clinical experience it has become possible with almost mathematical precision to predict the range of accommodation for any given age in emmetropic eyes, in which ametropia has been corrected and glasses have been worn for considerable periods of time.

In fact, through determining the ametropia and accommodation, the age of a person may be ascertained.

See also: No. 721, McFarland et al, 1939.

B. ANOMALIES

49. ALLEN, T. D.: *Anomalies of accommodation, frequency and variety*, Arch. Ophth., 4:84, 1930.

It has been demonstrated that anomalies of accommodation are frequent, occurring in 2 per cent of the cases in a general ophthalmic practice. The examples given are samples, not extremes, of more than 100 cases recently reviewed. By having a classification similar to one of those suggested, it will be easier to study the patient for the purpose of correcting or alleviating the condition.

50. COGAN, D. G.: *Accommodation and the autonomic nervous system*, Arch. Ophth., 18:739-66, 1937.

The sympathetic system tends to adapt the eye for relatively distant objects and, as such, opposes the parasympathetic system which tends to adapt the eye for relatively near objects.

How the sympathetic system effects this distance adjustment is not obvious. Probably the radial fibers of the ciliary muscle are innervated by the sympathetic system and on contraction exert a pull on the zonule which flattens the lens. They would be opposed, of course, by the circular fibers which on contraction decrease the circumference of the ciliary ring and allow greater curvature of the lens.

Reference is made to the evidence for dual innervation of the eye by analogy, as well as from the point of view of comparative anatomy, and also of anatomicophysiologic factors.

Sherrington's law of reciprocal innervation was confirmed by A. Bielschowsky's own investigations.

An increased capacity to accommodate was found in a series of 5 cases by the author, varying from 0.5 to 2.5 D., with marked contraction of the pupil on the same side, after surgical and other damage to the cervical portion of the sympathetic system.

See also: No. 371, Cooper, 1930.

C. ALTITUDE

See: altitude-anoxia—accommodation.

D. EXAMINATION

See: examination—accommodation.

E. FATIGUE

See: fatigue—accommodation.

F. SPEED OF ADJUSTMENT

51. FERREE, C. E. AND RAND, G.: *The speed of adjustment of the eye for clear seeing at different distances*, Tr. Am. Ophth. Soc., 16:142-63, 1918.

By "speed of adjustment" is meant here "speed in the action both of the extrinsic and intrinsic muscles in adjusting for clear seeing at different distances." In this particular, the authors believe that the aviator must excel.

In the study of this phenomenon three subdivisions have been made: the lag in the response of the retina to colored and colorless light, the lag in the adjustment of the eye for clear seeing at different distances, and the lag in the development of the perception of depth or distance. The relation of all of these to the functional efficiency of the eye is considered briefly.

The authors showed that as compared with its inertia of adjusting clearly to receive its impression, the inertia in its sensory reaction is relatively unimportant for most of the work which is required.

Both the adjustment of the eye and the clear seeing of objects are, in general, the important ocular functions involved in the judgment of distance. The testing of these extra-

ocular capacities, the ability to estimate and to learn to estimate distance is perhaps just as important as the testing of the ocular capacity itself. The testing of the ocular capacity as registered in certain simple space judgments, with or without the element of time or speed of performance, is capable of definite treatment.

The time required for the 18 normal observers to pass from near to far varied between 0.50-1.16 second, a range of 132 per cent; from far to near, between 0.39-0.82 second, a range of 110.3 per cent; and from near to far and back to near, from 0.96-1.76 seconds, a range of 83.3 per cent.

There is a possible bearing on the principles of the test on the work of the clinic, that is, if the conventional acuity test accuracy alone is taken into account. No provision is made in the form of the test to include speed of performance.

The results which the authors have obtained so far on older subjects suggest that above the age of 30 the eye becomes not only slower in its reactions but increasingly liable to lapses and depressions lasting through longer or shorter intervals of time which make it a tardy and unreliable instrument for much of the work it is called upon to do, especially for the special vocations requiring speed of performance.

The tachistoscope described was used in France for the study of the diurnal variations in the aviator's ocular fitness for his work. It has also been used by the Ophthalmological Division at the Medical Research Laboratory at Mineola, Long Island. Among the problems to be considered are: (1) the standardization of the test for the selection of aviators; (2) a study of the diurnal variations in the aviator's ocular fitness for his work, and (3) a study of the ocular effects of oxygen poverty.

52. FERREE, C. E. AND RAND, G.: *The inertia of adjustment of the eye for clear seeing at different distances. A study of ocular functions with special reference to aviation.* Am. J. Ophth., 1:764-76, 1918.

See Abstract No. 51.

53. FERREE, C. E. AND RAND, G.: *The speed of adjustment of the eye for clear seeing at different distances. A study of ocular functions with special reference to aviation.* Am. J. Psychol., 30:40-61, 1919.

Speed in the action of extrinsic and intrinsic muscles in adjusting for clear seeing at different distances is discussed. A careful check should be kept on the sensory and motor foundations of the fitness of aviators for their work. In this procedure the speed of reaction of their skeletal as well as their ocular muscles should be included.

An apparatus is described, which was used in France, and also by the Ophthalmic Division of the Medical Research Laboratory at Mineola, Long Island. This apparatus served for the study of the diurnal variations in the aviator's ocular fitness for his work.

54. FERREE, C. E. AND RAND, G.: *Human factor in airplane crashes.* Arch. Ophth., 18:789-95, 1937.

In considering airplane crashes, the pilot is an important factor. The authors believed that tests should be used to select pilots and thus reduce accidents. If pilots were tested before and after each flight, it would be possible to prevent a flight by a pilot unfit to fly at the time. These tests would also indicate the pilot's resistance to the strain of flying

and would offer the possibility of constructing curves for each pilot, indicating his endurance, susceptibility to fatigue, consistency, and other factors.

For this testing the authors designed an electric multiple exposure tachistoscope. A near object is exposed on the left, a far object in the median plane, and a near object at the right. The effect of fatigue upon speed of discrimination of objects and speed of accommodation may be tested. Thus, dynamic functional speed of vision, stressing either the oculomotor or the accommodative feature, may be evaluated, and the time taken to change from near to far and from far to near may be measured either in combination or separately.

55. GOODALL, E. B.: *The speed of accommodation.* Air Serv. Inform. Circ. 1 (3): 63-69, 1920.

The author used the apparatus designed by Ferree (see Abstract No. 51). Of 100 men examined, 89 were airplane pilots who had each spent from 15 to 500 hours in the air. Their ages varied from 21 to 44 years. The remaining 11 men were picked at random from among the workers of the laboratory.

The following averages show the characteristics of the groups tested:

Group	Description	Average Years	Near	Near to Far	Near to Far and Return	Outward Difference	Return Difference
I	73 aviators normal	24.2	0.030	0.844	1.429	0.815	0.584
II	17 aviators, initial delay	27.8	.121	1.186	1.796	1.065	.611
III	10 laboratory men and officers, with correction	31.8	.044	1.099	1.646	1.055	.547

In analyzing the first group it was found that the average time of the outward excursion of accommodation increased by 0.009 second for each year increase in age from 21 to 31 years. The coefficient of correlation in this case was 0.143 with a probable error of 0.077. No correlation was found between age and return time of accommodation. Nor was there in this group any correlation between outward time and return time.

Uncorrected astigmatic errors and conditions of muscle imbalance tended to retard the speed of adjustment to a marked degree.

In a few tests with older men, when attempting to estimate their speed of adjustment or accommodation, it was found that they had uncorrected hypermetropia or an appreciable presbyopia and were unable to distinguish near letters even with the widest sectors in the apparatus. In the case of several men having made poor time records, it was ascertained that they had but little sleep for several nights and, therefore, were below par physically. Three aviators had taken whisky a short time before the examination. Two made very poor records, while the third man's accommodative rate was so slow that no time record could be obtained.

56. LEUKART, R. H.: *The speed of monocular accommodation.* J. Exper. Psychol., 25:257-70, 1939.

The best solution to the problem of timing the reaction appears to be the objective regulation of the exposure time of the crucial object. The apparatus of Ferree and Rand (see Abstract No. 51) consists of a series of cut-out disks, revolved by an unbalanced pendulum,

so arranged that a near test object is seen, followed by a far one (or vice versa), and the exposure times of each can be controlled and varied. The "adjustment" with this apparatus involves convergence and change of lateral fixation, as well as accommodation to different distances.

Banister and Pollock measured far to near accommodation by using two fall-tachistoscopes. The large letters were exposed at 610 cm., the small ones at 41.5 cm. The ability to name the near letter after momentary exposure was the criterion of accommodation.

The author devised a method in which a change of fixation was not necessary while the accommodative movement was taking place. A modified Meumann time sense apparatus was used as an important part of this device. Only the star wheel was used in a vertical plane. On this a light cardboard disk was mounted, which revolved through 60 degrees at each contact of the two revolving arms.

On the disk were mounted two small rectangular mirrors, placed at slightly different angles to the observer's eye, so that one reflected the near object and the other the distant one to the same point. Another mirror was fastened to the edge of the table to the right of the observer's head at such an angle that the test objects were in position to the right and in front of the observer.

A black tube with a black screen mounted thereon was introduced between the observer's eye and the small mirrors on the star wheel.

The author draws the following conclusions: (1) In the technic used the action of the extra-ocular muscles (of the eye being used) apparently has been eliminated, since changes of fixation and convergence do not accompany the accommodation movement. Also, an objective criterion of accommodation has been incorporated into the device. This criterion, although apparently reasonably successful, can be improved upon.

(2) The results of the subjects used in the investigation indicate that in a group of persons the average accommodation time for the left eye is approximately equal to that for the right eye. For the distance at which the test objects were placed in this experiment (1 M. and 4 M.) there is no difference between the time required for near to far and for far to near accommodation.

(3) Inspection of the individual readings of right and left eye indicates that the fact there is no reliable difference in means for left eye and right eye is a function of probability.

(4) For far to near accommodation, 13 subjects (about 93 per cent) showed a difference in time for right and left eye of more than .060 seconds (the approximate unit of time measurement). For near to far accommodation, this same situation is observed in 50 per cent of the cases.

57. ROBERTSON, C. J.: *Measurement of the speed of adjustment of the eye to near and far vision*, U. S. Nav. M. Bull., 32:275-83, 1934.

The speed of vision and adjustment of the eye in seeing an object at 30 cm., a far object at 6 meters, and a second near object at 30 cm. may be determined by means of the Ferree-Rand tachistoscope. Ferree upheld that there are two types of tests for the speed of vision, a static and a dynamic test. The first, in which the eyes fix, is considered only suitable for testing the speed of reaction on the sensorium and would not test eyes with inferior oculomotor facility. In the dynamic test the eyes are required to shift from a near object on the left to a far object in the median plane and then return to a near object on

the right. Thus the first change involves relaxation of the muscles of accommodation and divergence and lateral movement to the right. The second change involves accommodation for near vision and a converging and a lateral movement still further to the right; constituting three factors which contribute to the final result: speed of reaction of the sensorium, speed of accommodation for near and far, and speed of coordinated eye movement. Only multiple examinations, including aviators of various ages, different refractive errors, muscular imbalance, etc., will show the value of the tachistoscope and give a standard of speed for a given age or speed of vision necessary for piloting certain types of planes. Goodall found that uncorrected astigmatic errors and conditions of muscle imbalance tend to retard the speed of adjustment. The tachistoscope is useful in determining defects of vision which are not readily revealed by Snellen's test letters. Richmond and Ebert, using flash letter tests, projection lantern and camera-box shutters, found that a time factor should be included in visual acuity examinations, that 0.5 second is a good standard unit for each eye, that visual speed by daylight is faster than by artificial light, that some candidates with slow color perception compensate with rapid form perception, and that speed of perception for both eyes is twice that of monocular vision.

Robertson obtained the following results for 62 subjects for total time (near to far and return to near):

Subjects	0.95-1.15	1.15-1.35	1.35-1.55	1.55-1.75	1.75-1.95
Aviators (30 to 38 years)	2	10	2	1	0
Midshipmen (16 to 24 years)	10	4	1	0	0
Unselected, no glasses (18 to 40 years)	3	5	8	1	1
Unselected, glasses (22 to 44 years)	0	1	3	7	3
Time in Seconds					

58. ROBERTSON, C. J.: *Measurement of speed of adjustment of eye to near and far vision.* Arch. Ophth., 14:82-89, 1935.

Speed of vision in aviation is essential and measurement of this speed can be made.

A standard of measurement of speed of vision can be established if sufficient tests are made on men of various ages. Such a standard, if established, should be incorporated in the visual requirements for aviators. Such tests disclose the pathologic eye.

59. ROBERTSON, C. J.: *A comparative study of the measurement of the speed of adjustment of the eye for near and far vision,* U. S. Nav. M. Bull., 33:187-205, 1935.

Using the Ferree-Rand multiple exposure tachistoscope, the results of tests for speed of adjustment for near and for far vision, covering the complete excursion times (near to far plus far to near), were as follows:

Age Years	Number	Pilots Average Time	Number	Nonfliers Average Time
15 to 19.95	—	—	16	1.357 seconds
20 to 24.9	17	1.244 seconds	49	1.288
25 to 29.9	93	1.218	25	1.326
30 to 34.9	99	1.348	14	1.341
35 to 39.9	34	1.396	1	2.13
40 to 44.9	6	1.639	—	—

Sixteen fliers who had longer excursion times than the majority of their age groups revealed inequality of vision, astigmatism, high exophoria at 33 cm., and hyperphoria. Of this group, 8 had had one crash and 2 had more than one.

Excursion times were found to be greater in 20 men with prism divergence of more than 8 diopters than in 20 men with divergence of from 2 to 7 D. This was more marked for near to far than for far to near.

In a comparison of two groups, each including 33 men, it was found that the group with exophoria at 33 cm. of 10 D. or more were slower in all phases than the group with 5 D. or less of exophoria at 33 cm. The younger pilots did not show this difference as markedly.

It was shown that speed of adjustment is slowed up by muscular imbalances, inequality of vision, refractive errors, and other visual factors. The author believed that below the age of 30 years a speed greater than 1.35 seconds should be disqualifying for pilots, a speed above 1.55 seconds should be disqualifying at ages 30 to 40, and a speed greater than 1.75 seconds should be the upper limit for all ages. Below 30 years of age the limit for near to far adjustment should be 0.90 second, and above 30 the limit should be 1.0 second. Fat to near limit below 30 should be 0.096 second, and over 30 years of age it should be 0.70 second.

In the tests the lighting was continuously checked with the Macbeth illuminometer (under an illumination of 5 foot candles). A tachometer was used to check the motor. It is considered that exophorics have more trouble in converging and take a longer time in passing from far to near (the slowness is even more marked as age increases)—while those with esophoria have more trouble in diverging and take longer to pass from near to far. It was noted in the tests that those found to have visual errors of any type are slower in their performance on the tachistoscope. Age has a marked bearing on speed of adjustment of the eye to clear seeing for different distances. With any anomalous eye condition the age factor becomes of greater importance in the speed of adjustment. After 30 years of age the changes become rapidly more pronounced. Speed of adjustment of far to near is decidedly faster in all ages than near to far. Inequality of visual acuity of the eyes, not necessarily below the required 20/20 vision, has a definite bearing on the speed of adjustment of the eyes. Data on depth perception are insufficient as yet to make definite statements. A standard of speed of adjustment through measurement can and should be accomplished. The pathologic eye is disclosed through tests on the tachistoscope.

60. Robertson, C. J.: *Measurement of speed of adjustment of eye to near and far vision. A further study.* Arch. Ophth., 15:423-34, 1936.

Age has a marked bearing on the speed of adjustment of the eye to clear seeing for different distances. With anomalous conditions of the eye the age factor becomes of greater importance in the speed of adjustment. After the age of 30 the changes in the speed rapidly become more marked.

The following conditions definitely cause a slowing of the speed of adjustment of the eye to near and far vision: age, exophoria of 8 diopters or more at 33 cm., astigmatic errors in one or both eyes, vision below 20/20 in one or both eyes, inequality of vision even though above the required 20/20, myopia, a low angle of convergence, low or unequal accommodation, hyperphoria, esophoria of 6 diopters at 6 meters associated with hyperphoria at the extreme limits, and sluggish reaction.

It is considered that the premises which were made originally have been upheld, that speed of adjustment is essential in aviation, that a measurement of such speed can be made, that a standard should be established, and that the pathologic eye can be demonstrated with tests with the tachistoscope.

61. Robertson, C. J.: *Effect of fatigue on the adjustment of the eye to near and far vision*, Arch. Ophth., 17:859-76, 1937.

This report adds to the previously reported measurements with the tachistoscope of the speed of adjustment of the eye to near and far vision. The data on tests of dynamic speed of vision for pilots with fatigue after 1, 2, 3, and 4 hours of flying are tabulated and compared with the normal standards. Age and various anomalous conditions of the eye have marked bearing on the speed of adjustment at all ages, but more marked as age advances.

62. Tefft, L. E. and Stark, E. K.: *The speed of accommodation as a practicable test for fliers*, Air Serv. Inform. Circ. 4 (359):25-27, 1922, and Am. J. Ophth., 5:339-42, 1922.

Working with the tachistoscope of Ferree, Tefft and Stark studied the speed of accommodation to ascertain if it would provide a practicable test for aviators. Three test figures were exposed at a distance of 6 meters and immediately afterward 3 at a distance of 30 cm. The interval between the exposures was reduced to the lowest that allowed the subject just to discriminate, so that he could give a majority of correct judgments. The time required varied from 1.2 to 2 seconds. They conclude that such a test is not especially valuable for fliers because those who failed in it would be disqualified by other conditions more easily ascertainable.

See also: No. 421, Kirchhoff, 1940; No. 638, Ferree & Rand, 1938; No. 856, Ferree & Rand, 1939.

IV. Aeroembolism

See: decompression.

V. Age. Effect of, on the Eyes

A. ACCOMMODATION

(See also: accommodation-age)

See: No. 48, Duane, 1912; No. 51, Ferree & Rand, 1918; No. 55, Goodall, 1920; No. 59, Robertson, 1935; No. 60, Robertson, 1936; No. 61, Robertson, 1937; No. 721 McFarland et al, 1939.

B. DARK ADAPTATION

See: light sense—dark adaptation.

C. DEPTH PERCEPTION

63. Pol, W.: *Inferior distance judgment in old pilots (Sur l'évaluation inférieure de distance chez les pilotes âgés)*, Polska Przegl. Med. Lotn., 6:34-36, 1937. Abstract: J. Aviation Med., 9:77, (March) 1938.

The author made a study of distance judgment in 29 pilots who had been dismissed

from military service because of impaired distance judgment but who were still permitted to fly. One hears often at aviation schools of pupils who have been refused owing to poor landings, in spite of an excellent certificate from the oculist.

The writer divided his 29 pilots into two groups: The first group comprised 10 subjects with a visual acuity of both eyes not exceeding 0.8. Only 1 of these men was perfectly healthy, 1 had syphilis, another was psychologically unfit, 2 had advanced neurosis, 5 hypertrophy of the thyroid gland and strabismus of varying degree.

In the second group 19 pilots showed a difference in acuity of the two eyes. Four showed strabismus of different degree. The visual acuity of the affected eye varied from 0.3 to 0.7 owing to hypermetropia or astigmatism. Some pilots wore correcting glasses, which did not always improve their evaluation of distance.

Such a marked reduction in visual acuity has not developed during service. There must have been hypermetropic astigmatism or hypermetropia before. Only a few cases of myopia had developed during service.

The factors contributing to good distance evaluation include good eye function, especially perfect visual acuity, equal refraction of both eyes, absence of apparent or latent strabismus, a wide visual field, and psychic factors for conjugation of ocular impressions. In cases of inferior distance evaluation one or more of these factors are absent, whether ocular or psychic or both. The question arises whether pilots with very slight ocular deficiencies should be admitted. It is hard to disqualify a pilot who has thousands of flying hours behind him merely because of a slight defect in vision. It must be kept in mind that such defects rarely remain stationary, but progress and may sooner or later lead to catastrophe.

D. ILLUMINATION

64. Ferree, C. E., Rand, G., and Lewis, E. F.: *Age as an important factor in the amount of light needed by the eye.* Arch. Ophth., 13:212-26, 1935.

Determinations of visual acuity made on a group of presbyopic and nonpresbyopic eyes at light intensities from 0.5 to 100 foot candles are discussed.

The size of the pupil affects the clearness of the image, as follows: the breadth of the cross section of the cone of light from each point of the object is changed; the effect of diffraction on the clearness of the image is changed; and aberration and other irregularities of focusing, resulting from the use of the periphery of the lens and cornea, are excluded by pupils of small and medium size.

With advancing age, changes in density occur which are unequally distributed throughout the lens. By decreasing the size of the diffusion circles in the image and by limiting the aperture to the most favorable portions of the refracting system, the pupil acts as a final corrective of all defects of focusing.

Reduction in the size of the pupil exerts a secondary effect on the clearness of the image. In passing to the retina a certain percentage of light is scattered by internal reflections within the media.

These are, besides others (such as the inferior imaging power of the refracting media with its diminished transparency, and the decay of the processes of adaptation and adjustment), the reasons why an old eye needs more light.

E. INTRA-OCULAR PRESSURE

65. GRADLE, H. S.: *The effects of mydriatics upon intra-ocular tension.* Am. J. Ophth., 19:37, 1936.

Gradle has shown that tension after the use of a mydriatic increases in direct proportion with age. He found that the curve of pressure according to age is fairly uniform, being lowest in the 30 to 40 decade, rising steadily to a maximum in the 60 to 70 decade, and decreasing after the age of 70.

F. MISCELLANEOUS EFFECTS

66. BEACH, S. J.: *Senile changes and the eye examination.* J. Aviation Med., 3:9-12, 1932.

Since forerunners of the most common senile changes are frequently found in the eye, the examiner may be able to estimate the patient's resistance to the effects of age.

Failing distant vision, glaucoma, vascular degenerations and records are discussed.

67. GORE, T. L.: *The effect of age on flying.* J. Aviation Med., 5:80-95, 1934.

The physical effects of age have a definite bearing on the progress of commercial as well as military and naval aviation. The author pointed out the great value of close application to the protection of the physical fitness of the flier in promoting his longevity.

A great increase in functional neuroses of the gastro-intestinal (5 per cent a year) and circulatory system is observed in aviators; which is more often temporary, but leaving indelibly on the individual a greater predisposition to the occurrence of organic diseases.

Most conspicuously is this illustrated in the increased incidence of alleged ideopathic or essential hypertension individuals who come into the hypertension group and who, in the case of aviators in general, come into it 5 or 10 years earlier than that usually expected. This abnormal "aging" is due to the occupation and not to the inherent character of the pilot. Even after prolonged rest and treatment his condition may fail to correct itself, in which case the inevitable organic manifestations, following persistent hypertension, occur more quickly.

In time of peace the minimum age of the student pilot has been fixed at 20 years; after the age of 24 one learns flying more slowly.

The best age for combat leadership is between the ages of 27 and 35. Above 35, if still active in combat flying, the aviator is too much the "old timer" to teach other individuals.

At about 40 there is a disintegration incident to age, but the better type of human being does not start on the downward path until 45. After that age rapid physical disintegration is found in many military pilots. Their eyesight is the most apparent of these physical disintegrations.

Frequently nervous symptoms are produced by the fear that their physical disintegration is harming their flying ability to the point where they will be removed as pilots.

68. WILMER, W. H.: *Diseases of the eye in old age.* Arch. Ophth., 1:42-62, 1929.

The effects of disease in the young simulate the degenerative changes of age. In the ocular abiotrophies, certain tissues present at birth resemble the appearance of age, or they may have implanted in them the seeds of premature senility. In youth, a wasting disease may cause one to look "hollow-eyed," with malposition of the eyelids; there is an arcus juvenilis as well as an arcus senilis; dystrophies of the cornea change its curvature, translucency, and luster. Cataracts may be congenital, or they may occur early in life from nutritive or toxic disturbances of the lens. Infection may cause uveal and fundus changes not unlike those that are called senile. Inflammations also induce the disintegration of the pigment epithelial cells of the iris, with atrophy and migration of the pigment granules. Glaucoma may be congenital from a fault in tissue development, or it may follow intrauterine inflammation. The senile condition, "retinitis atrophicans centralis," is simulated in youth by traumatic or inflammatory lesions of the choroid and retina (so-called "hole in the retina").

See also No. 322, Sillevaerts, 1936; No. 324, Brouwer and Jongbloed, 1940; No. 900, Hargreaves, 1938.

VI. Airsickness

69. FLACK, M.: *Air-sickness and sea sickness.* Proc. Roy. Soc. Med., United Services Sect., 24:635-641, 1930-31.

In airsickness the conflict between visual, labyrinthine, and deep muscle sensations is a disturbing factor. However, this is lessened by giving passengers as wide a view of the horizon as possible.

70. YANQUELL, C. C.: *Airsickness.* U. S. Nav. M. Bull., 37:486-89, 1939.

Eye muscle imbalance and effort syndrome can simulate the symptoms of airsickness. True airsickness is a normal condition to which every normal person is susceptible and which nearly always can be overcome by habituation to flying. Supposed intractable cases almost invariably have a pathologic base and must be so diagnosed. From the etiologic side, the designation of vestibular hyperstimulation might be more appropriate for airsickness. The aerial maneuver causing airsickness most readily is the snap roll which is a violent 360 degree rotation of the plane about its horizontal axis while in level flight. The Immelmann turn is next in frequency (a half roll and 180 degree change of direction upon the summit of a loop).

For airsickness, sedatives are contra-indicated in an occupation requiring alertness and acute judgment. Sodium amytal (3 grains) appears to be the most reliable drug. The wearing of an abdominal binder is justified.

In one case marked ocular muscle imbalance and double vision was found in a naval aviator of 10 years' experience. He eventually retired because of this physical defect which could not be remedied sufficiently to make him safe for aviation.

See also: No. 5, von Diringshofen, 1939; No. 309, Fulton, 1940; No. 322, Sillevaerts, 1936; No. 1042, Friedländer, 1913; No. 1047, Hirschlaff, 1918.

A. THERAPEUTICS

See: therapeutics—air sickness.

VII. Alcohol

71. LOTTIG, H.: *On the influence of alcohol, nicotine and lack of sleep on efficiency at high altitudes (Ueber den Einfluss von Alkohol, Nikotin und Schlafmangel auf die Höhenfestigkeit).* Luftfahrtmed. Abh., 2:218-233, 1938. Abstract: J. Aviation Med., 10:62, 1939.

Alcohol and tobacco in large quantities should be avoided by aviators. A moderate indulgence is not harmful if sufficient sleep is obtained. Lack of sleep per se has a more harmful effect. A combination of all three must be avoided. Aviators must be warned that they are likely to overestimate their own efficiency because of the absence of effect or insignificant effect on single performance capacity. The changes are manifest in finer psychic reactions which the aviator himself is incapable of recognizing or evaluating.

The findings show that detrimental effects of alcohol and nicotine are slight as regards altitude capacity provided sufficient sleep is obtained. (Similar conclusions were drawn in 1918 in the Medical Research Laboratory at Mitchell Field, C. B.)

72. MILIAN, M. G.: *Aviators and alcohol (Aviateurs et alcohol).* Bull. Acad. de méd., Paris, 121:636-38, 1939.

Milian asked the question: Why select the personnel by so rigid an examination of reflexes, nervous system, etc., when aviators are allowed to drink a quantity of alcohol sufficient to annihilate all these capacities?

According to the author, excessive consumption of alcohol is a habitual and socially necessary convention among aviators.

The average monthly consumption by officers in one aviation camp was: five liters of vin-apéritif (port and pernod); one liter of spirits or brandy; one liter of liqueurs; one liter of beer or wine a day. Non-commissioned officers consumed approximately one-fourth more than officers.

73. POWELL, W. H.: *Ocular manifestations of alcohol and a consideration of individual variations in seven cases studied.* J. Aviation Med., 9:97, (June) 1938.

Alcohol, being a coagulant of protein, tends to irritate and destroy cells, thereby being a general protoplasmic poison.

In orthophoric individuals a small amount of alcohol produces gradually increasing esophoria; larger doses provoke esotropia with diplopia in the primary position.

Alcohol causes gradual loss in prism divergence and decrease in the angle of convergence due to diminished muscle tonus and muscular control. Finally, a complete loss of power of convergence may be the consequence. However, it has practically no influence on visual acuity.

If alcohol interferes with binocular fixation, the manifestations become significant.

See also: No. 55, Goodall, 1920; No. 78, Tanaka, 1929; No. 125, Wright, 1929; No. 288, Fombeure, 1931; No. 915, Neuberger, 1922.

A. ALTITUDE-ANOXIA

See: altitude-anoxia—alcohol.

B. TOBACCO-ALCOHOL AMBLYOPIA

See: tobacco-amblyopia (tobacco-alcohol).

(To be continued)

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ABSTRACTS

ophthalmology

DIAGNOSTIC METHODS OF EXAMINATION, BIOMICROSCOPY AND PHOTOGRAPHY

The Effect of the Suction Cup Sucked to the Surface of the Eye on Intraocular Pressure and the Serviceableness of the Suction Cup as an Ophthalmodynamometer. R. SYSI. Helsinki, Finland. Acta. ophth. *28*:261-70. Fasc. 2. 1950.

The possibility of using the suction cup as an ophthalmodynamometer was investigated, using the eyes of pigs with eyelids and orbital fat in situ. The measurements were compared with those obtained by means of a mercury manometer inside the eye. It was found that the rise in pressure inside the eye differed markedly from the negative pressure of the suction cup. The pressure of the eye did not rise evenly but in jerks and in different ways in different tests. The shape and size of the suction cup were important factors in producing different pressures with the same suction. Even the weight of the suction tube may have caused considerable changes in the intraocular pressure.

It is concluded that the suction cup is valueless as an ophthalmodynamometer as it introduces too many sources of error. 12 references, 3 figures.—*J. Zuckerman.*

The Retinal Mirror Reflexes. EJLER HOLM. Copenhagen, Denmark. Acta ophth. *28*:23-32. 1950.

The retinal reflexes were examined to obtain an impression of the retinal surface. The examination was performed with a dilated pupil and erect picture. If the pupil is contracted and the picture inverted, isolated reflexes such as the circumfoveal reflex become stronger and more distinct. When the mirror is moved in front of the pupil, the reflexes, except for those from the macular region, arise from almost plane surfaces. If elevations or depressions are present, the reflexes become more persistent. The central reflexes may be called foveolar and circumfoveal reflexes. The foveolar reflexes are formed when a concave mirror is moved in a direction opposite to the movements of the eye and sometimes yields an inverted picture of this mirror as it is seen with the electric ophthalmoscope with the mirror before the lower half of the hole. The hole in the mirror may account for the reflexes that have previously been described as circular. The reflex has been described as lying entirely before the fovea,

particularly in binocular ophthalmoscopes and in stereoscopic photographs. This is due to the fact that two images are formed in different parts of the retina. In the same way reflexes appear, on a concave surface, to lie behind the surface.

The circumfoveal reflexes of the two eyes can only be compared when the pupils are of equal size. This reflex is of clinical importance because it becomes irregular, broken, or "wrinkled" in edema of this area. The visual acuity may, however, not be disturbed because the foveola is not affected. A similar appearance has been found in sympathetic ophthalmia and in iridocyclitis. It has also been observed in two young patients with early diabetic retinitis.

The author considers the tunica media to be the most important reflecting medium. In retinitis of hypertension the reflexes are increased because hyaline degeneration is present and they are reflected from a greater part of the artery so that the reflexes are less sharply limited and the cylindric shape of the artery becomes visible (copper-wire artery).—*J. Zuckerman.*

A Simplified Astigmometer. JAMES E. LEBENSOHN. Chicago, Ill. Arch. Ophth. *43*:905-907, May, 1950.

A vinylite plastic chart has been devised for testing astigmatism. It consists of two long lines which constitute the cross and two short lines which form an arrowhead for locating the axis. All the lines are 5 mm. in width. If the clearest line is vertical, the arrow is pointed to 90 degrees; if it is horizontal, to 180 degrees. If one wing is blacker, the arrow is rotated away from the blacker wing until matching takes place. The shaft of the arrow will then be at the correct axis.

The axes are indicated with dual numbering completely around the dial. The outer figures are read when the dial is viewed in the mirror; the inner figures (in parentheses) are noted when they are viewed directly. The use of the dial in conjunction with a mirror facilitates the examination.

After the correction is determined, the eye is fogged by 0.25 D. and the exact axis verified by the Crisp-Stine method, using the arrowhead figure on the original Crisp-Stine test. To simplify the latter alternative, a small triangle of black Scotch tape is placed at the midpoint of the arc subtending a clear quadrant. With the marker placed at the axis under consideration, the comparisons are made between the arrow line and the plain line. In cases of low astigmatism, it is often helpful to check one method by means of the other. 1 reference.—*J. Zuckerman.*

Effects of Uniform and Non-Uniform Surrounds on Foveal Vision. GLENN A. FRY. Columbus, Ohio. Am. J. Optom. & Arch. Am. Acad. Optom. *27*:423-36, Sept., 1950.

An analysis of the results of experimental studies on the effects of uniform and nonuniform surrounds on foveal vision reveals that objects in the periphery of the field of vision may affect foveal vision in several ways:

1. Bright or dark objects in the periphery may cause distraction which interferes with speed and efficiency in performing a visual task requiring foveal vision.

2. A bright peripheral object may influence foveal vision indirectly as a result of its effect on the size of the pupil.

3. If the field of view is not uniform and the eye is permitted to move from one part of the field to another, the eye may be required to perform a critical visual task after the fovea has been adapted to a high level of luminance.

4. A bright source in the periphery can cast stray light on the foveal image at the fovea.

Other factors to be considered are the possibility of diffusion of a substance from one part of the retina to another, the effect of electrical potentials generated by stimulating one part of the retina upon the activity initiated in another part of the retina, and interaction at synapses.

It is concluded that restricting the surroundings of a test object to an area of 5 degrees in diameter is satisfactory for use both in investigation of visibility of test objects and in the study of visual performance. It is also satisfactory for the routine clinical measurement of visual acuity.—*J. Zuckerman.*

OCULAR MOVEMENTS AND MOTOR ANOMALIES, NYSTAGMUS, READING DISABILITY

Balancing Visual Stimuli in Orthoptic Training. MATTHEW LUCKIESH, R. M. HALL AND S. K. GUTH. Cleveland, Ohio. Am. J. Optom. *27*:3-7, Jan., 1950.

The authors describe a new technic for controlling the visibility of objects viewed through orthoptic instruments by simultaneously changing the brightness and contrast of the target. A circular colorless filter with a precise gradient of density is placed before the dominant eye and adjusted until suppression of that eye occurs and fixation is transferred to the squinting or nondominant eye. Binocular vision is attained by adjusting (1) the size of the targets, (2) the amounts of prismatic power, or (3) the density of the filter before the dominant eye. Training is then conducted in the ordinary manner. As the visual efficiency of the nondominant eye increases, the density of the filter before the dominant eye is progressively reduced.

The technic has been used extensively in clinical practice and has proved to be effective in obtaining single binocular vision in many cases which failed to respond to other methods of control. 5 references. 3 figures.—*Author's abstract.*

Relation of Refractive Error to Optometric Tests. LEO MANAS. Chicago. Optometric Weekly: 1473-76, Oct. 5, 1950.

A statistical study was made of the effect of refractive error upon the (no. 15A) phoria obtained through the dissociated cross-cylinder finding at near in 768 cases. From his data, the author concluded that the low coefficient of correlation found indicates no significant relationship between refractive error and the (no. 15A) phoria. —*Robert E. Bannon.*

A Comparison of Clinical Methods of Measuring Accommodative-Convergence. MEREDITH W. MORGAN. Berkeley, Calif. Am. J. Optom. & Arch. Am. Acad. Optom. 27:385-96, Aug., 1950.

The Maddox concept of the components of convergence is briefly discussed, namely, that convergence may be divided into: tonic, accommodative, proximal, and fusional convergence.

The four most common methods of measuring accommodative convergence are: (1) the gradient method, (2) the phoria method, (3) the cross cylinder/phoria method, and (4) the relative accommodations/phoria method. In the first method two near phorias are used. Immediately after the near phoria is taken, a second one through a 1.0 D.S. add is taken. The difference in the two phorias represents the change in convergence per unit of accommodation. In the second method the accommodative convergence is calculated from the fixation distance, P. D., distance phoria and near phoria. This method includes proximal convergence as well as accommodative. The last two methods of measurement are not accurate.

The values obtained from clinical measurements of accommodative convergence by the gradient and the phoria methods on 413 subjects were compared. The mean value by the gradient method was 9.1Δ and for the phoria method 12.6Δ. The difference between the two values, 3.5Δ, represents proximal convergence.

The regression lines indicate that proximal convergence is a special function of fusional convergence and that it is probably a conditioned reflex rather than an innate one.—*Author's abstract.*

Pre-Orthoptic Care of the Very Young Squinters. WILLIAM SMITH. Boston, Mass. Am. J. Optom. & Arch. Am. Acad. Optom. 27:397-403, Aug., 1950.

In this paper a system of simple preventive-corrective steps for pre-orthoptic care of the very young squinter is presented. The basis for the procedure is: (1) optical correction when needed, (2) prolonged occlusion of the nonsquinting eye, (3) intensified performance of visual tasks with the squinting eye.

Importance of early care of strabismus is stressed. Emphasis is laid on the danger of deterioration of perceptual functions if treatment is delayed. The author points out that the extent and intensity of deterioration is in relation to age at which squint occurs.

Several types of occluders are mentioned and described, as well as the author's own method of occlusion. Occlusion in general is discussed and the historical background of the practice is reviewed.

A routine of clinical tests which can be given to very young children is presented, with occlusion serving as an important medium for evaluating the child's seeing ability. This method is valuable in cases where it is not possible to determine acuity by conventional means. Smith recommends skiascopy as the objective test from which optical correction may be prescribed. Subjective verification is to be done only if possible. Observation, case history, and such objective tests as are practicable are ample for the beginning. 9 references.—*Author's abstract.*

Orthoptic Training in Convergence Deficiency Uncovers Latent Vertical Imbalance—A Case Report. HOWARD F. HAINES. Columbus, Ohio. Am. J. Optom. & Arch. Am. Acad. Optom. *27*:357-60, July, 1950.

A high school student, aged 20, apparently in good health, was referred by his reading counselor for visual help as the result of a school survey indicating poor fusion. Refraction indicated:

O.D.—0.50 D. Sph = —0.25 D. Cyl Axis 120. V.A. = 20/20
O.S.—1.75 D. Sph. V.A. = 20/20
Phorias at 6 M. Vertical O, Lateral 1Δ exophoria
Phorias at 40 CM. 11Δ exophoria

Normal vertical balance was also indicated by stereoscopic test cards used with the Keystone Telebinocular.

Convergence training was instituted on the Rotoscope and the Brock Stereomotivator with Keystone overhead projector, using a large plastic intermediary translucent screen. Fusion was difficult and stereopsis poor. A vertical imbalance began to become manifest. As this developed, it was temporarily compensated for with fitovers until a total of 2Δ of right hyperphoria was measured. This was accompanied by marked improvement in fusion and stereopsis. Incorporated in the lens prescription was 2Δ of vertical prism, normal stereopsis was developed, and the near point phoria was reduced to 7Δ to 9Δ of exophoria, with normal, positive convergence reserve.—*Author's abstract.*

Paralysis of Extraocular Muscles following Spinal Anesthesia. H. C. TSENG AND Y. C. KU. Peking, China. Chinese M. J. *68*:81-84, March-April, 1950.

In a period of six years the authors encountered only 2 cases of paralysis of ocular muscles following spinal anesthesia, which was employed 2,132 times during that interval. Both cases occurred after appendectomy. The patients were males, aged 32 and 51, respectively. Total dose of procaine hydrochloride used was 150 mg. for the first patient and 480 mg. (in divided doses for continuous spinal anesthesia) for the second. The blood pressure was maintained at the patient's original level by subcutaneous injections of ephedrine, although in the second case it fell from 200 mm. Hg. to 90 mm. Hg. for a short time. Postoperatively, the patients complained of severe headache for a few days. Diplopia occurred on the sixth and fifth days after operation, respectively. Examination of the eyes revealed bilateral paralysis of the abducens nerve in the first case and paralysis of the right oculomotor nerve in the second. No other abnormal neurologic findings were obtained.

Large doses of yeast orally and vitamin B_1 parenterally were given. In the first case the paralysis of extra-ocular muscles greatly improved about three months after its onset, but diplopia was still present four and one half years after the operation when looking in an extreme lateral direction toward either side. In the second case the diplopia completely disappeared about three weeks after its occurrence.

The cause of ocular complications following spinal anesthesia was not clear. The author did not attach any definite causal significance to the long-standing chronic paranasal sinusitis which the first patient had, but he thought that in the second case the presence of hypertension and the marked fall of blood pressure during the spinal anesthesia might be considered a possible factor in the production of paralysis of extra-ocular muscles by affecting the cerebral circulation. 11 references.—*H. L. Chang.*

CORNEA, SCLERA AND TENON'S CAPSULE

Corneal Transplantation. MAURICE CROLL AND LEO CROLL. Detroit, Mich. J. Michigan M. Soc. *49*:442-49, April, 1950.

Four types of corneal transplants are reported:

1. *Total Corneal Transplant.*—This type, which involves the entire cornea, has not given any measure of success.

2. *Total Lamellar Transplant.*—The entire upper half of the cornea, extending from limbus to limbus (approximately 0.5 mm.), is removed and replaced with the same size graft. The European school of keratoplasty reports good success.

3. *Partial Lamellar Transplant.*—This is similar in technic to 2, except that the size of the graft does not extend to the limbus (usually 5.0, 6.0, 7.0 mm., etc.). It is advocated by the European School because of a large measure of success.

4. *Partial Penetrating Transplant.*—This involves the transference of a complete corneal disc from donor to host, varying in size (4.5, 5.0, 5.5, 6.5, 7.0 mm., etc.). This type is the most universally used because of the high percentage of success.

All cases should be thoroughly studied from all aspects, rechecked, and, in the final analysis, each case should be considered strictly on an individual basis.

From the standpoint of prognosis, all cases fall roughly into three large groups:

Group 1. Favorable, includes (a) keratoconus, (b) inactive interstitial keratitis, (c) central scars, (d) other criteria including no vascularization, active infection, or increase of intra-ocular pressure.

Group 2. Partially Favorable, consists of large number of corneal dystrophies with two exceptions, i.e., Fuchs's dystrophy, fatty dystrophies.

Group 3. Unfavorable, is an heterogeneous, miscellaneous group consisting of (a) eyes with nystagmus, (b) chemical burns, (c) corneal scars extending to limbus, (d) large degree of vascularization of cornea, (e) inflammations of uvea, (f) dystrophia adiposa, and (g) Fuchs's dystrophy.

The technic is largely an individual one depending on the operator. In planning the operation, the following considerations must be established preoperatively, namely:

Round or square graft.—Cosmetically round graft is preferred and also is easier to perform. Visual results are the same in both types.

Bevel or not.—Donor graft and host are cut with the same trephine. Because of curvature of cornea, some bevel is always present.

Size of graft.—This depends on the amount of involved corneal tissue to be resected. The tendency today is toward larger size grafts (7.0, 8.0 mm.). There is no contraindication to large grafts, except that iris incarceration must be guarded against.

Sutures.—All grafts require sutures, either overlying or direct. A triple triangular overlying suture in combination with direct sutures in the area not covered is advocated.

Routinely, a 1 ml. incision is made outside of the graft into healthy host cornea to a depth of 0.3 mm., since this trough stops invasion of superficial vessels postoperatively. No ointments are used after the operation.

Complications may be encountered during or after surgery:

1. *Placing of incision.*—If not properly placed, trephine is removed and replaced to cover required area with no untoward effect.

2. *Perforation into anterior chamber.*—This is encountered at the time of inserting overlying sutures. Withdraw suture and place superficially into cornea.

3. *Lens injury.*—If lens is injured in young person (soft nucleus), it will absorb. Treat conservatively. If it occurs in older person (hard nucleus), remove lens at time of operation, leaving in posterior capsule to prevent herniation of vitreous into anterior chamber.

4. *Anterior synechiae.*—This is prevented by operating on eye with undilated, small pupil and inserting atropine (to dilate) just before graft is put into place.

5. *Removal of sutures.*—This is done usually between 10 and 12 days. There is a danger of collapse of the anterior chamber. If this occurs, insert a direct suture at the point of weakness, or treat conservatively since graft will reattach.

6. *Removal of anterior synechiae.*—Anterior synechiae always cause opacification of the graft. In a minimal adhesion a closed eye procedure is done, as in cyclodialysis a spatula is inserted into the anterior chamber and synechiae are separated followed by air injection. In large synechiae the eye must be opened and a large iridectomy done. Surgery should be done only after the fourteenth day.

7. *Glaucoma.*—Treat glaucoma as any postoperative complication. If it is a congestive type, do a large broad base iridectomy; for the noncongestive type which persists, do a trephine or iridencleisis.

8. *Edema.*—There is no specific treatment for edema, but it usually disappears within a six month period.

Five cases of corneal transplantation with resulting vision, followed for from 12 to 30 months, are reported with pre- and postoperative photographs.—*Author's abstract.*

Chickenpox in the Cornea. EMIL FRANDSEN. Kolding, Denmark. Acta ophth. *28*:113-20, 1950.

After a brief survey of the cases of keratitis associated with chickenpox reported in the literature, description is given of 2 additional cases. The 2 patients were children in whom a universal eruption of chickenpox was followed, about 10 days later, by a

deep central infiltration of the corneal parenchyma, characterized by being sharply defined with a distinct, peripherally more dense marginal zone. In one case the lesion took a protracted course, with ingrowth of deep vessels and development of typical keratitis disciformis, and it took five to six months before the affection healed, with a large opacity remaining in the cornea and 6/24 vision. In the other case the keratitis healed in two weeks, leaving only little opacity, with 3/6 vision.

The late appearance of keratitis is an interesting feature which, in analogy with the late complications of the central nervous system, is interpreted to the effect that the lesion is due to an allergic reaction in the corneal parenchyma.

It is further pointed out that in chickenpox two types of keratitis may develop: (1) the late parenchymatous form here described, and (2) a superficial form that appears simultaneously with, or shortly after, the exanthema. The latter is due to vesiculation of the cornea. Often it has a phlyctenular appearance and takes a rapid though mild course. 25 references.—*Author's abstract.*

A Case of Primary Band-Shaped Opacity of the Cornea. JENS EDMUND. Copenhagen, Denmark. Acta ophth. *28*:209-14, Fasc. 2, 1950.

Most of the cases of the band-shaped opacity of the cornea described are of the secondary form, while genuine primary cases are extremely rare. Nevertheless, the first case published (Dixon, 1848) belongs to this group.

This patient is an 80 year old woman. On both corneas, situated in the interpalpebral area, a symmetric, slightly yellowish brown, dense, band-shaped opacity is noticed. Microscopically it is situated just below Bowman's membrane. The thickness of the cornea is not increased; there are no corneal vessels. Sensibility is slightly reduced in the affected part. The remaining part of the cornea is normal. Neither on the cornea nor in the remaining part of the eye is any sign of present or previous inflammatory changes found. In the anamneis as well as in the general examination it has been impossible to find any feature of importance concerning the corneal lesion or affording an explanation of it.

The typical clinical picture is described and the histologic picture discussed. It is presumably a question of accumulation of hyaline material in the superficial part of Bowman's membrane (Verhoeff), probably intracellularly (McCallan), which later calcifies.

The former theories are mentioned of the primary form being preglaumatous (v. Graefe), a manifestation of uric diathesis (Nettleship), a result of changes in the corneal vessels (Leber), or a disturbance of the corneal nutrition (Sisson, Chan a.o.). In favor of this, the change found in Bowman's membrane very much resembles the change of elastic tissue in the wall of the vessels in arteriosclerosis.

According to this, the author regards the lesion as a progressive sclerosis of the same character as the general sclerosis originating from elastic tissue. It is a senile hyaline change of the very cells belonging to the tissue and not an intercellular accumulation of foreign material, in this way differing from the infiltrative degeneration of the cornea. 23 references.—*Author's abstract.*

Influence of Donor-Recipient Sensitization on Corneal Grafts. A. E. MAUMENEE. Palo Alto, Calif. Presented at the Nineteenth Scientific Meeting of the Association for Research in Ophthalmology, Inc., June 25, 1950.

The clouding of mechanically successful corneal grafts three to five weeks postoperatively has not, heretofore, been explained adequately in an eye which would be expected to support a clear transplant. It is well known that most tissues of the body when transplanted will "take" and survive for three weeks, but that they are eventually destroyed by donor-recipient sensitization. Clear control grafts do not participate in this type of reaction. The reason for this may be that the comparatively small number of cells transferred in a corneal graft do not produce sufficient antigen to cause a sensitization, and the relatively avascular site of the recipient cornea is a poor bed for that type of reaction. It seems reasonable, however, that if almost every other tissue in the body produces a sensitization when transplanted from one individual to another, corneal transplants would at least occasionally produce a similar response. To determine if the clarity of a corneal graft could be influenced by donor-recipient sensitization, the dose of the antigen was increased by inserting a piece of skin from the original donor under the panniculus carnosus of the recipient two weeks after a corneal graft had been performed. Twenty-eight clear grafts in 30 animals so treated became cloudy two to four weeks after the insertion of the skin transplant. This clouding of the grafts was in striking contrast to the late clouding of only one graft out of 60 animals in which a booster skin graft was not given. The clinical appearance of the ocular reaction in the sensitized animals was similar to that observed in the late clouding of corneal grafts in man. Histologic sections of the grafts which became cloudy as a result of sensitization in rabbits showed an inflammatory reaction similar to that which occurs in homogenous skin grafts. Cortisone delayed the clouding of corneal grafts which was produced by sensitization. Not enough experiments have been done to determine whether this medication can prevent the clouding of corneal grafts, but it seems theoretically possible that it may do so, for the biologic individuality of corneal transplants appears to last for only six to eight weeks after corneal grafting.—*Author's abstract.*

ANTERIOR CHAMBER & PUPIL

Functional Unilateral Mydriasis. Report of Three Cases. JOSEPH ROSS. Berwick, Pa. Arch. Ophth. *43*:823-33, May, 1950.

The anatomy and basic physiology of the pupillary pathways and the clinically important pupillary reflexes were reviewed. Three cases were presented of functional unilateral mydriasis occurring in young, healthy, white, married but nonpregnant, myopic women who did not wear glasses. Vision in all 3 was unaffected, but the pupils were immobile to light and in accommodation, and reacted but slightly in convergence. The condition in these cases resembled somewhat an aberrant type of Adie's syndrome, and the usual studies revealed no clue to the cause. All the patients recovered in 48 hours. Pupillary studies gave interesting, but not helpful, results. A

possible site of the lesion in the inferior, efferent (constrictor) branch of the third cranial nerve, just after this branch leaves that to the inferior oblique muscle, was suggested. Such a lesion would involve the light reflex and the near reflex (accommodation and convergence) and might conceivably leave the convergence element of the near reflex less affected. If the fibers for accommodation and convergence are separate and those for accommodation leave after those for convergence, near the ciliary ganglion, then a single lesion at the point of departure of these fibers could produce the picture observed in these cases. Central lesions might also be present in such cases and supplement the peripheral derangement. The relative rapidity of onset and recrudescence suggests a vascular cause, possibly a vasomotor phenomenon of chemical origin, conceivably related to an allergic disturbance. The reason for the unilaterality is not clear. The syndrome was so named for want of a better appellation, even though none of the patients appeared to be psychoneurotic. Such cases probably appear oftener than the literature indicates and are of more academic than practical interest, since in none of the present cases have any permanent sequels, ocular or otherwise, occurred during at least two years' observation, but, admittedly, this interval is too short to be sure such occurrences do no presage serious central nervous system, ocular, or other disease.—*Author's abstract.*

UVEAL TRACT AND SYMPATHETIC OPHTHALMITIS

About Anterior Choroiditis in Chronic Uveitis. A. HUGGERT. Stockholm, Sweden. Acta Ophth. *28*:121-27, 1950.

It is generally supposed that anterior choroiditis is caused by lues, tuberculosis, sympathetic ophthalmia, or lepra. The author has collected material containing 23 cases of chronic uveitis with an anterior choroiditis in which the etiology was neither lues nor sympathetic ophthalmia. In 7 of the cases etiology was benign lymphogranulomatosis verified by biopsy; in 4 it was probably the same, while in the remaining cases it was uncertain. Fresh choroiditic patches were found in one half of the cases; nodules in the iris tissue often occurred at the same time. It is the author's opinion that anterior choroiditis is common in chronic uveitis, but that it is seldom found as the examination of the peripheral parts of the fundus oculi is difficult. The importance of indirect ophthalmoscopy at this examination is emphasized.—*J. S. Guyton.*

RETINA

Placental Implants in Ophthalmology. S. ETZINE. Johannesburg, South Africa. M. J. *24*:681-83, Aug. 19, 1950.

Wald's work on the identification of carotenoids in the eye of *Rhesus pipiens* stimulated Josephson and Freiburger to give intramuscular injections of carotene in oil to patients with retinitis pigmentosa. Filatov confirmed the favorable results claimed by these workers, but failure of the same form of therapy by oral administration led him

to believe that disintegration products of the liver were in question. Filatov's work on corneal grafts tended to show that, when tissues such as cornea, placenta, or skin were stored at a low temperature, they formed "biogenic stimulants." He was therefore curious to know whether such biogenic stimulants would produce a similar effect to the disintegration products of the liver. He implanted placenta subconjunctivally in 22 patients with retinitis pigmentosa and claimed that all were improved.

These remarkable claims led to the method being tried in many of the Western European clinics. In this paper the results from French, Italian, Swiss, and other clinics are reported and analyzed. Most workers agree that placental implants produce an improvement in some cases of retinitis pigmentosa, but that this is never very great. The patients frequently feel better, but objective changes are not visible. Several implants may be required to produce an effect, which then lasts four to six months.

Details are also given of the results claimed in high myopia with complications, where an improvement in the distortion of images and in vitreous opacities has been described. The improvement in certain corneal lesions described by some workers using the same form of therapy is also noted.

The author discusses the difficulties in making an evaluation of the claims made for this form of therapy. 23 references.—*Author's abstract.*

Electrophysiological Studies of Color-Blindness. KOITI MOTOKAWA AND KITIRO SUZUKI. Sendai, Japan. Tohoku J. Exper. Med. 52:195-206, May, 1950.

The electrical excitability of the eye after two seconds' illumination was investigated by applying a single, constant, current pulse of 0.1 second to the eye through a pair of silver electrodes placed on the forehead above the eyebrow and on the homolateral temple. The "excitability curve," obtained by plotting percentage increases in electrical excitability as ordinates against the time after pre-illumination as abscissas, showed a single maximum at about two seconds in normal man; while the curve obtained from deuteranomalous subjects showed three distinct elevations which were termed "R," "G," and "B" in the order of rapidity of development. It was shown that the three elevations represent three independent processes, not different phases of one and the same process. By further elaboration of the measurements, it has recently been revealed that there is another independent process, "Y," which has an intermediate crest time between R and G and is considered as mediating the sensation of yellow. In this respect, the retinal processes at the fovea of deuteranomalous subjects resemble those found in parafoveal regions of normal men. The wave length dependence or spectral locus of each color process was investigated. The locus of the B process has its maximum at about 460 mμ in normal and color-blind subjects. The R locus was found very weak or lacking in protanopia and the G locus in deuteranopia. The locations of their maxima were 610 mμ and 530 mμ for R and G, respectively, showing agreement with the result obtained from parafoveal regions of normal man. For further detail, see Ebe, Isobe, and Motokawa: Science, 1951, in press. 6 references, 6 figures.—*Author's abstract.*

Hemangioblastoma of the Retina (Von Hippel-Lindau Disease). ANDREW RADOS. Newark, N. J. Arch. Ophth. *43*:43-65, Jan., 1950; 265-329, Feb., 1950.

Reviewed in great detail are the very earliest observations, historical data, and clinical appearance of angiomatosis retinae (Von Hippel-Lindau disease).

Two cases are reported in which the author had an opportunity to examine slides of the eyes. The first case was that of a girl, aged 16, and the second that a 28 year old man. In both cases the tumors were intraretinal and of the capillary angioma type, with endothelial proliferation and sproutings, and the presence of cystic cavities. In the first case the subretinal transudate was extremely rich in cholesterol crystals; in the second the number of these crystals was negligible, but the transudate exhibited a large number of swollen, desquamated, and proliferating pigment epithelium derivatives corresponding to the ghost cells. In the second case the hyperplasia of the connective tissue elements between the capillary loops was extremely marked.

In the section on patho-anatomic observations the author notes the great confusion that exists in the early literature on the clinical and pathologic evaluation of the vascular growths. Lindau was the first to recognize that angiomatosis of the retina is a part of a syndrome, and there is a correlation of tumors of the central nervous system and those of the retina, due to underlying early maldevelopment and their coordination with malformations or tumors of the somatic organs.

Von Hippel-Lindau disease is distinctly of hereditary character and, in summarizing the data on the hereditary factor of the disease, the author points out that horizontal, vertical, horizontal-vertical, and collateral involvement occur. The hereditary involvement in the vertical direction was demonstrated in a much larger group than the horizontal inheritance. The largest group of pedigrees revealed horizontal and vertical inheritance in families in which the members were affected either with manifestations of the eyes or with those of the central nervous system or with both simultaneously. It is concluded that the inheritance is doubtlessly a pleiotropic monogenic variety affecting the tissues of mesodermal origin, dominant in character. The gene is autosomal, no sex linkage being present in the different pedigrees.

The author devotes the final sections of his paper to differentiation between hemangioblastoma of the retina and similar conditions, such as retinitis exudativa (Coats's disease) and angioma retinae. The two latter conditions have no hereditary basis. —*Author's abstract.*

NEURO-OPHTHALMOLOGY, OPTIC NERVE, VISUAL PATHWAYS, CENTERS AND VISUAL FIELDS

Syphilitic Optic Neuritis. G. S. GRAVESON. Manchester, England. J. Neurol., Neurosurg. & Psychiat. *13*:216-24, Aug., 1950.

The paper is based on a study of 5 cases of this rare and ill-defined condition. In 1 patient the optic neuritis occurred in the secondary stage of infection; in the other 4, in the teritary period. Two of the patients were man and wife, and the neuritis occurred in them simultaneously.

Full clinical details, including visual field charts, are given in each case. From these it has been possible to define the clinical pattern of the lesion more precisely. It starts, like the common acute retrobulbar neuritis, with sudden visual failure in one eye, but in all cases seen at this stage a *bilateral* papillitis was present. In 4 cases the discs were intensely hyperemic, with blurred margins but no gross edema or hemorrhages. In the fifth an enormous edema of the nerve head was seen together with a periphlebitis and local choroiditis near the disc head.

The characteristic field changes in the early stages were (1) an enlargement of the blind spots, and (2) concentric constriction of the smaller isopters. These changes were found in eyes opposite to those subjectively affected. Visual failure results when fibers are more severely damaged, and at this stage central or paracentral scotomata and nerve bundle defects may be found.

The Wassermann test was positive in the blood in all cases and also positive in the cerebrospinal fluid in the 2 cases which showed fluid changes.

In marked contrast to primary syphilitic optic atrophy, the prognosis of optic neuritis is very good if adequate treatment is given early. All 5 patients improved satisfactorily on penicillin therapy.

The differentiation of the condition from the diseases producing optic neuritis and from other syphilitic disorders producing disc changes is discussed.

The scanty literature on the subject is reviewed and the lack of adequate pathologic study is stressed. The clinical observations suggest, however, that initially the disease is a papillitis, and that from this point the inflammation may spread either into the retina and choroid or backward along the optic nerve. On this basis it is possible to group together such conditions as syphilitic neuroretinitis, choroiditis juxta papillaris, perioptic neuritis, Fuchs's neuritis papulosa, etc., as being varying manifestations of essentially the same underlying inflammatory disturbance. 7 references, 10 figures.—*Author's abstract.*

GLAUCOMA AND HYPOTONY

Postoperative Gonioscopic Evaluation of Pressure Reducing Operations in Simple Glaucoma (Postoperative gonioskopische Untersuchungen drucksenkender Eingriße bei Glaucoma simplex). E. G. A. VAN BEUNINGEN. Berlin, Germany. Arch. f. Ophth. *149*:620-36, Heft 6, 1949.

Postoperative gonioscopic examinations were made following 146 operations in 115 patients suffering from simple glaucoma. It was demonstrated that the size of the chamber is not the determining factor, but rather the functional capacity of the trabecular network and Schlemm's canal in the region of the coloboma. The various factors responsible for the success of the various operations, including Elliot's trephination, cyclodialysis and iridectomy, are discussed. In the first mentioned intervention it is emphasized that, in contradistinction to the statement of some American writers, closure is favored by prolapse of the pedicle of the coloboma and ciliary processes and withdrawal of the lens into the trephination aperture. In cyclo-

dialysis it is not only the width of the angle that determines success but chiefly the technic of the operation itself. In 62 cases of Elliot's trephination no definite reduction in pressure could be demonstrated in spite of medical treatment. In 58 per cent of these cases, the operation was successful. Another gonioscopic proof of the dangers of the Elliot trephination is the relatively small number of eyes which will respond to medical treatment if the fistula fails. Whereas 30 per cent of cases in which iridectomy was performed could be regulated by medical treatment, less than 6 per cent of the cases in which the Elliot operation was used could be thus regulated. In 46 patients operated on by the inverse trephination cyclodialysis of Heine-Blakovics, ocular pressure could not be regulated medically in 10 cases.

In 5 cases the cyclodialysis was done following iridectomy, but pressure could not be regulated due to pigmentary obstruction of Schlemm's canal and adhesions of the tunnel. In contradistinction to Goldmann, the present writer is not of the opinion that, in the presence of a narrow angle, a preliminary iridectomy is necessary before proceeding to cyclodialysis, even though a wider angle does yield better results. The use of a heavier spatula in cyclodialysis is recommended as presenting less danger of hemorrhage and less danger of closure of the tunnel. It also yields better results in the presence of a narrow angle.

In comparing the end results of the three types of operation, the best results were obtained with cyclodialysis in which a successful outcome was noted in 68 per cent of the cases. Then followed the Elliot trephination with 58 per cent of successful results and, last, iridectomy with 52 per cent. Since the difference is so small, it cannot be stated that statistics prove the superiority of any one of the procedures, as regards the pressure-reducing effect. 40 references, 3 tables.

No American writer has been so foolish as to claim that inclusion of ciliary processes and the crystalline lens in the filtration channel insures success. Almost all eyes are lost when such extensive inclusions prevail.—B. F. P.

Glaucoma—Surgical Complications. PHILIP M. LEWIS. Memphis, Tenn. J. Tennessee M. A. *43*:44-47, Feb., 1950.

Iridectomy.—Iridectomy is used principally for narrow angle glaucoma in the acute stage or in the period immediately following the acute attack. As the anterior chamber is often very shallow, it is difficult and dangerous to make the incision either with a keratome or with a cataract knife. The layers of the cornea may be split with the keratome so that the final entry is too far anterior to the iris root. The lens capsule may be injured with the point of the keratome on entering the anterior chamber or on withdrawing it. During the latter maneuver, if the point of the keratome is tilted too far forward in an effort to avoid the lens, the corneal endothelium may be seriously damaged. On entry, the keratome point may engage the iris and cause iridodialysis, hemorrhage, or both.

To avoid these complications, which should never occur unless the anterior chamber is very shallow, the incision should be made *ab externo*, after turning down a small

conjunctival flap. This is a relatively easy and safe procedure and requires only a few minutes more time than the usual keratome incision.

Such serious complications as lens dislocations, vitreous loss, and total iridodialysis are almost always due to the patient's misbehavior, which is due to the surgeon's failure in preparing him properly. General anesthesia, preferably pentothal sodium, may be necessary if the patient is very nervous and unruly and the eye acutely inflamed. However, in most cases the surgery of acute glaucoma can be performed safely under local anesthesia by sufficient preoperative sedation plus ample retrobulbar and subconjunctival injections of procaine with epinephrine.

Corneoscleral Trephination.—A complication that may sometimes occur during operation is buttonholing or tearing the conjunctival flap. Occasionally it can be closed successfully with a 6-0 purse-string suture. If, as is usually the case, the hole is just at the corneoscleral junction, closure is difficult. Unless the trephine opening has already been made, it is wise to abandon the original position and do the operation to one side or even below. If the trephine opening has been made and the hole cannot be closed properly with sutures, the following procedure is necessary: Complete the operation as usual, do the iridectomy, and suture the conjunctival flap and Tenon's capsule. Then dissect the conjunctiva cleanly from the limbus on either side of the trephine opening, thus preparing a Van Lint sliding flap. With a knife or a small sharp curet remove the epithelium from the cornea just below the trephine opening and horizontally across the limbus on each side. Then suture the conjunctival flap to the episcleral tissues of the limbus just distal to the 11 and 1 o'clock positions.

Intra-ocular hemorrhage usually stops spontaneously and should be irrigated out. Scleral bleeding is controlled by touching with the actual cautery or diathermy.

The trephine blade must be sharp and held properly at a right angle to the limbus; otherwise the button of sclera may be cut through imperfectly and its entire removal made very difficult, resulting in early closure of the opening. It is desirable to put no instrument whatever into the trephine opening unless absolutely necessary.

Vitreous loss is a very serious complication, usually preventing successful filtration so that the operation is a failure. It should be prevented by not trephining too far posteriorly and by avoiding undue pressure and roughness in operating.

Postoperative complications are (1) iritis, (2) failure of the anterior chamber to reform, (3) failure of filtration, (4) cataract formation, (5) hypotony, (6) prolapse of iris, and (7) endophthalmitis or panophthalmitis. These complications are listed in order of frequency in which they have occurred in my experience. Prolapse of the ciliary body is not listed because I have never diagnosed it. It undoubtedly has occurred in some cases in which the operation failed.

(1) Iritis has occurred rather frequently, requiring the use of neosynephrine, heat, and milk injections in addition to atropine. The patient should remain in the hospital as long as there is a definite iritis. Usually it subsides within 10 days.

(2) I like the anterior chamber to restore on the third day. It worries me if it has not reformed on the fifth day. If it has not restored on the seventh day, an injection of air should be done. This may be done rather easily by the method of Paul Chand-

ler, without removing the patient from his bed. When the anterior chamber stays flat too long, adhesion of iris and/or lens to cornea, adhesions of iris to lens, and rapid cataract formation will occur.

(3) Failure of filtration means failure of the operation. It may be due to fibrotic closure of the scleral opening, prolapse of ciliary processes into the opening, or dense adhesions of the conjunctiva to the opening and the surrounding sclera. While some have recorded good results from dissecting down the flap and reopening the wound, in my hands this has failed due to the formation of even more scar tissue.

(4) Any glaucoma operation seems to hasten the formation of cataract in elderly patients. It seems to me that it occurs most frequently in trephines.

(5) In my opinion hypotony is not serious, and I have never had a case that showed marked degeneration from it, although several have had a pressure below 12 mm. for years. In most cases no treatment is indicated. Two patients who have had one hypotonous eye and one with normal tension have been followed for a number of years. In both cases the soft eye has maintained the better vision.

(6) In my experience prolapse of the iris after operation is very rare. Should it occur, it should be dealt with promptly by dissecting down the conjunctival flap and excision of a large section of iris.

(7) I have encountered 2 cases of panophthalmitis, 2 and 6 years respectively after the trephination, both requiring evisceration. In a much larger number of iridencleses I have had only 1 case of ophthalmitis which developed seven years after a successful operation. While I have not had the opportunity, it seems possible that, if a case of endophthalmitis were seen in an early stage, intensive treatment with modern chemotherapy and antibiotics might check the infection before a hopeless panophthalmitis developed. Irrigation of the anterior chamber with penicillin or streptomycin should be tried and probably intravitreal injections.

Iridencleisis.—There are few complications which occur at the time of operation. Injury to the lens and corneal endothelium can occur in cases with shallow anterior chambers, but this can be avoided by making the incision *ab externo*.

Hemorrhage into the anterior chamber is rarely severe and should be removed by irrigation. Scleral bleeding should be checked by touching with the cautery. Occasionally an iris pillar may slip back into the anterior chamber, but if drawn well into the wound angle, it will not occur. If it happens, great care must be taken in regrasping it with forceps because the lens may be injured.

Complications occurring postoperatively are (1) failure of the anterior chamber to reform, (2) closure of the wound with complete failure of filtration, (3) cataract formation, (4) hypotony with excessive bleb formation, (5) panophthalmitis, and (6) sympathetic ophthalmia.

(1) Usually the anterior chamber has reformed by the third or fourth postoperative day. If it has not reformed in one week, it constitutes, in my opinion, a serious complication. Permanent central opacity of the cornea and rapid formation of cataract are likely to occur. Injection of air by the method of Chandler will restore the anterior chamber by pushing the lens and iris back from the cornea.

(2) Closure of the wound and failure of all filtration results in some cases. Early massage tends to prevent it. In some cases in which there is no apparent filtration the tension can be maintained within normal levels by the use of miotics postoperatively, when they had no effect preoperatively. In most cases, however, reoperation of some type will be necessary.

(3) Cataract formation may occur, but, in my experience, has been less frequent than after trephination. Failure of the anterior chamber to reform quickly is a potent source of lens clouding.

(4) Hypotony, with excessively large blebs, occurred in both eyes of one of my patients (Kodochrome shown). This is a rare development. One of these blebs became so large as to seriously interfere with vision and comfort. It was also very disfiguring. Surgical removal was done three years after the iridencleisis operation. A conjunctival flap was dissected and brought down to cover the denuded area. Vision improved and the eye became comfortable. For a short while the pressure rose to 35 mm. and required miotics, but soon filtration resumed and the tension fell to 12 mm. and later 10 mm. On increase in the size of the bleb recurred and required further surgery.

(5) Panophthalmitis is a very rare complication of iridencleisis. I have had one case occurring seven years after a perfectly successful operation. When seen one week after the inflammation began, it was impossible to determine by history or examination whether the panophthalmitis developed from a conjunctivitis which spread through the scleral opening or whether it was a metastatic infection.

(6) Sympathetic ophthalmia has been reported,[1] but fortunately this dreadful complication is exceedingly rare. I have never had a case and feel that the remote possibility of its occurrence should not deter one from performing this most valuable operation for glaucoma.

Cyclodialysis.—This operation is used by the writer only for the relief of increased intra-ocular pressure in aphakia. In this condition it is the operation of choice.

The principal complications at the time of operation are (1) hemorrhage into the anterior chamber, (2) perforation of the choroid, and (3) injury to the endothelium and Descemet's membrane.

(1) Hemorrhage into the anterior chamber may be brisk and serious. The injection of air, as recommended by Barkan and also by Randolph is valuable in its control. Pressure with a firmly rolled, moist cotton sponge over the sclera, with the spatula in position, as suggested by O'Brien, is also useful in checking hemorrhage.

(2) Perforation of the choroid in making the scleral incision should never happen. If the incision is made with the "flat" of the knife and slanted anteriorly so as to bevel the incision, there is no danger of perforation. If the spatula "hugs" the sclera during its passage into the anterior chamber, it will not tear the choroid nor injure the ciliary body. In one case in which I injected air, the choroid was perforated with the tip of the gold canula and air was injected into the vitreous. Fortunately no serious damage resulted.

(3) Injury to the endothelium and to Descemet's membrane will occur if the tip of the spatula is allowed to scrape against the back of the cornea. This may produce a permanent opacity of that area. To avoid this, the spatula should not be thrust too

far or too vigorously into the anterior chamber.

Complications occurring after operation are few, if any, except for failure to lower the pressure. This is due to closure of the cleft. Avoidance of trauma with the spatula, plus the injection of air, with positioning of the head postoperatively (Barkan) makes closure of the cleft less frequent and the operation more often successful. The daily use of miotics is also helpful. If closure occurs, the operation may be repeated in another quadrant or even at the same site.

Cyclodiathermy.—I have used diathermy of the ciliary body a total of 17 times, following the technic of Dunphy and Albaugh. Good results have been reported by Stocker and others from penetrating diathermy, but I have never employed it, probably because of undue fear of causing hemorrhage or sympathetic ophthalmitis. My comments will therefore be confined to the nonperforating procedure. I consider it especially valuable in uncontrolled glaucoma secondary to uveitis.

The complications at the time of operation are (1) the difficulty of dissecting the conjunctival flap in cases having had several previous operations, (2) occasionally a very marked increase of tension requiring immediate posterior sclerotomy. The postoperative complications are the result of (1) overcoagulation and (2) undercoagulation. The first may be disastrous; the latter requires reoperation.—*Author's abstract.*

Good and practical.—B.F.P.

Atropine as a Pressure-Reducing Agent in Juvenile Glaucoma. (Atropinals drucksenkendes Mittel bei kindlichen Glaukom). H. REMKY. Klin. Mbl. Augenh. *115*:539-43, Heft 5, 1949.

Having observed in 2 cases of embryotoxon posterius that the normal intra-ocular pressure diminished following instillation of a 1 per cent atropine solution, the author suggests the use of atropine as an antiglaucomatous agent in cases of dysgenesis mesodermalis with pathologically increased intra-ocular pressure. This treatment was tested in 11 young patients with glaucoma. The atropine drops contained atropine sulfate in 0.1 to 0.25 per cent solution. In 10 of these patients the intra-ocular pressure diminished following instillation of the atropine drops, but in 1 case this reduction in pressure was only transitory. Five patients were treated for several months and showed a return to normal intra-ocular pressure not obtainable by other drugs. Even long after treatment had been discontinued, 1 patient showed no recurrence, so that the possibility of a lasting cure is not excluded.

The cause of the pressure reduction following the use of atropine is believed to be a change in the vegetative factors involved in production of aqueous humor. The use of atropine is suggested for stationary patients in all forms of glaucoma associated with malformation of the anterior segment of the eye seen chiefly in children and very young patients. A detailed description of the 11 cases is included. 10 references.

Provocative tests should be performed before the patient is permitted to use atropine at home.—B. F. P.

EYELIDS

Granuloma Arteficiale in the Region of the Eye. R. SYSI. Helsinki, Finland. Acta ophth. *28*:257-60, Fasc. 2, 1950.

The author reports 2 cases of granuloma arteficiale in the eyelids. In the first case some "wound powder" containing talc had been introduced into the wound. Talc also occurred in the tumor which had formed, and thus its origin could be considered beyond doubt. The origin of the second case, on the other hand, remains obscure. At operation no foreign substance was introduced into the wound, as far as could be determined. Yet it may be supposed that some talc might have entered the wound from the operator's gloves, though none could be demonstrated in the histologic specimen. 5 references.—*Author's abstract.*

Gummas of the Eyelid. J. V. CASSADY. South Bend, Ind. Am. J. Ophth. *33*:18-22, Jan., 1950.

In earlier textbooks and ophthalmic literature gummas of the eyelid were relatively common. Tertiary luetic lesions are now so infrequent that we rarely consider syphilis of the eyelid. The similarity between gumma of the lid and multiple chalazion and the unusualness of the former may cause the diagnosis to be missed.

A review of the literature of gummas of the eyelids and the report of a case is presented. The case reported had been treated as multiple chalazion of the eyelid until the serology and a biopsy established the diagnosis as gumma of the eyelid. Induration of the eyelid and several black crusted ulcers superimposed upon blepharitis and ectropion of the lower lid was caused by gummatous induration of the eyelids.

Tertiary syphilis of the eyelid should be thought of if chalazions fail to heal, especially if ulceration onto the skin side of the lid is associated with them. 18 references. 1 figure.—*Author's abstract.*

ORBIT

The Use of Paladon as a Heteroplastic Implantation Material in Bony Defects of the Orbit and Its Vicinity. (Die Verwendung von Paladon als heterotopisches Implantations-material bei Knochendefekten im Bereich der Orbita und deren Umgeben.) E. WALSER. Munchen, Germany. Arch. f. Ophth. *149*:355-63, 1949.

In defects around the orbit, cartilage or bone implants will usually bridge the defect and not fill it. Dense adhesion of these substances to the skin favors cicatricial contractions, possibly leading to entropion. Cartilage shrinks, and both bone and cartilage easily lead to suppuration. Cartilage in this region acts as a foreign body. Paladon is a pink plastic material used by dentists. It is very hard, resistant to blows, has a low specific weight, is a poor heat conductor, and is chemically indifferent. It is easily molded and friendly to the tissues. The skin remains movable over it, and the implant does not change its shape. This material is recommended for implantation in all cases in which soft tissue surgery will not suffice, particularly for cases in which

the bony orbital margin has been destroyed. The cosmetic and functional effect is excellent. Before implantation it is imperative to exclude fistular suppuration or chronic foci of irritation in the vicinity of the defect, and there must be sufficient skin available to cover the implant. Four cases are described in detail with the technic of implantation. Previous to operation, 1 cc. of weak scopolamine-eukodal-ephetonine is injected intramuscularly and 10 cc. of clauden to prevent hemorrhage. A local anesthetic is used. The implant must not be introduced until hemostasis is perfect. After it has been put in place, the skin is sutured over it. The wound is covered with silver foil and a compression bandage is applied, which can be loosened the next day. The dressing is changed on the third day and, with careful attention to suture suppuration, can be removed after six to eight days. The implant is slightly movable at first but becomes fixed as cicatrization progresses. With smooth healing the patient can be discharged 10 to 14 days after operation. 8 references, 8 figures.—*S. Rodman Irvine.*

Nuerinoma of the Orbit. BRYNJULV STANDAL. University Hospital, Oslo, Norway. Acta ophth. *28*:49-70, 1950.

Neurinomas are forms of tumor which doubtless occur more frequently than was formerly assumed. They arise from the supporting tissues of the peripheral nerves, and most authors believe that the starting point for the tumor development lies in the cells of Schwann's sheaths.

Many neurinomas are purely innocent tumors, but they may sometimes be more malignant than was formerly believed. Incomplete extirpation is said to lead to malignant development, and radical surgery is therefore recommended. When the tumors have become malignant, the histologic atypia soon becomes more marked. The tumors may then be difficult to recognize and are frequently mistaken for spindle cell sarcomas or myxosarcomas.

The neurinomas usually occur as solitary tumors but are not infrequently multiple, and then often associated with a general neurofibromatosis. They are usually found to have definite localizations and are rarely seen outside thereof.

For ophthalmologists it is an important fact that neurinomas may occur in the region of the eye. They have hitherto been relatively little mentioned in ophthalmologic literature and only about 40 such tumors have been described. They are sometimes found in the eyelid, sometimes intrabulbarly in the uvea and retina, but most often as extrabulbar tumors of the orbit.

It has been shown that neurinomas of the orbit may originate from the ciliary nerves, from the oculomotor, and the supraorbital nerve. According to F. Davis, they cannot arise from the optic nerve, which lacks the Schwann's sheaths; whereas R. Müller believes that they can arise from the small nerves in the sheaths of the opticus and thus be closely associated with the optic nerve. The orbital neurinomas have a tendency to produce defects in the bone around the orbit and to penetrate into some of the sinuses and the cranium. Otherwise they present no special symptomatology.

The author has gone through the case records of the patients who have been treated for unilateral exophthalmus in the eye department of Oslo University Hospital during

the last 10 years. In 32 cases in which operations were performed the exophthalmus was due to primary orbital tumors, and histologic examination showed that 8 of these were neurinomas, which thus constituted 25 per cent of the total number and formed the largest group of orbital tumors operated on in this period.

In the same period one tumor was found arising from the inner side of the upper eyelid. This tumor, which was histologically malignant, had grown very rapidly and at the time admission hung far down on the cheek. The tumor was extirpated under local anesthesia, with good cosmetic and functional result and without signs of recurrence after two years.

Two of the orbital neurinomas were found on trial excision and histologic examination to be malignant. In both cases evisceration of the orbit was performed. In one case the patient was free from recurrence after 5½ years. In the other the tumor was, through a defect in the orbit, firmly adherent to the meninges and could not be radically removed. In this case the patient died seven months after operation. This was the only case with a fatal result.

The remaining 6 neurinomas showed no histologic signs of malignancy. In 2 of these cases the tumor was completely extirpated by Krönlein's operation. In two other cases the tumor could not be completely removed, as it had penetrated through a defect in the bone into the anterior cranial fossa, and in the last 2 cases the surgeon did not succeed in complete extirpation of the tumor.

In the last 4 cases there should, therefore, be a danger of malignant development. Hitherto no such development has been noted after an observation time of respectively 3½, 2, 1, and 10 years.

A remarkable feature in this material is the young age of the patients. The average age was 15.2 years, whereas neurinomas are otherwise reported to occur most frequently in middle life.

No signs of neurofibromatosis were observed in any of these patients or in their nearest relatives. 17 references, 4 figures.—*Author's abstract.*

The Present Status of Orbital Implants. J. S. CRAWFORD, Toronto, Canada. Canad. M. A. J. *63*:46-48, July, 1950.

A good cosmetic result following enucleation entails a normally filled orbit and full motility of the prosthesis. Good motility can be assured if the implant and prosthesis are made to interlock. There are essentially three such types of implants. The basket implant of Cutler illustrates the simplest type. It is buried, and when the socket is healed, a depression is formed. A conical stud is moulded on the posterior surface of the artificial eye to fit this depression. The range of motion is improved by using the ball and ring implant of Cutler, allowing direct contact between the implant and prosthesis. If the ring is replaced by tantalum mesh, more permanent results are obtained. Two implants of similar principle are described. The body of the implant is made of plastic and covered with tantalum mesh for muscle attachment. Operative procedures for evisceration, enucleation, and old sockets are described.

Conjunctival discharge with all types of exposed integrated implants is greater

than with buried or with no implants. The operative procedure is more involved than with buried implants. However, the cosmetic result is better and when the tantalum mesh implant is used, there is little chance of extrusion. 7 references, 2 tables.—*Author's abstract.*

Clinical Orbitonometry. J. H. MEANS and J. STANBURY. Boston, Mass. Am. J. M. Sc. *220*:357-61, Oct., 1950.

Perhaps the most poorly understood aspect of Graves' disease is the eye component, and this is especially true when the disease assumes that form which has been called the hyperophthalmopathic type wherein the eye picture dominates the situation. Here is described the use of a new instrument known as the Orbitonometer, recently introduced by Copper. The instrument measures the resistance of the globe to backwards displacement under increments of pressure applied. It is shown that the instrument gives stable readings. It gives promise of demonstrating in individual patients the course which the disease may take. 8 references, 4 figures.—*Author's abstract.*

PHARMACOLOGY, TOXICOLOGY AND THERAPEUTICS

Some Remarks on Retinal Vein Thrombosis and Its Treatment with Anticoagulants. SVEN LARSSON AND BORJE NORD. Lund, Sweden. Acta ophth. *28*:187-201, Fasc. 2, 1950.

In the Eye Clinic in Lund, Sweden, all cases of retinal vein thrombosis have been treated since 1938 with heparin and since 1945 also with dicumarol. Dosage of heparin was 250 mg. twice daily for 10 to 21 days. The dicumarol dosage is controlled every day so that the prothrombin index does not pass 40-50. One hundred patients were investigated: of 59 cases of complete thrombosis, 23 per cent improved: of 41 cases of tributary thrombosis, 58.06 per cent improved. Figures do not permit any definite conclusion as to the superiority of the method.

	Final Vision	No. Cases	Mean Age	No. Cases Blood Press. Normal	Initial Vision
Complete thrombosis					
Group 1.	0.9-1.0	11	36.2 yrs.	10	0.52
Group 2.	0.4-0.8	10	62.9 yrs.	2	0.41
Group 3.	1/60-0.3	23	55.3 yrs.	5	0.12
Group 4.	0-0.5/60	15	66.3 yrs.	2	0.1
Tributary thrombosis					
Group 1.	0.9-1.0	15	55.3 yrs.	0	0.46
Group 2.	0.4-0.8	13	60.5 yrs.	5	0.3
Group 3.	1/60-0.3	13	60.84 yrs.	2	0.17

Group 1 among the complete thrombosis stands out: Mean age was 36.2 years; there was a rise of blood pressure in 1 case out of 11; initial vision was 0.52. In this group are no ocular symptoms of angiosclerosis, no or little alteration of the intraocular tension, and general health is good, in contrast to all other groups. Is the pathogenesis here entirely different? Is an angiospasm the essential thing? (Transitory obscurations are often seen as precursors.) Anyway, materials containing this prog-

nostically favorable group do not prove the real value of any form of therapy, here anticoagulants.

Nine out of 44 complete thrombosis cases turned out finally as chronic primary glaucoma of *both* eyes, and 3 out of 34 tributary thrombosis cases were followed by the same complication (2 cases bilateral and 1 case in the *other* eye). Is there a common cause of the vascular thrombosis and simple chronic glaucoma, i.e., is the latter a vascular catastrophe too?

The authors believe that treatment with anticoagulants has not revolutionized the therapy of retinal vein thrombosis, but, from a purely theoretical point of view, it is very tempting, after all. The administration is, however, very difficult. Is this the reason for the rather discouraging results? 38 references, 8 tables.—*Author's abstract.*

Clinical Observations on the Use of Cortisone in Ophthalmic Diseases: Preliminary Report. JOHN W. HENDERSON AND ROBERT W. HOLLENHORST. Rochester, Minn. Proc. Staff Meet., Mayo Clin. 25:459-62, Aug. 2, 1950.

From October 1949 to May 1950 the authors had an opportunity to observe the effects of the adrenal cortical hormone, cortisone, on 8 patients who had various ocular diseases. In 4 of these cases a definite association between the ocular disorder and rheumatoid arthritis seemed to exist. In 2 of the patients the ocular disease was not associated with arthritis. Two cases of vernal conjunctivitis in children also were studied.

Among the cases in whom the ocular disease seemed to be part of an arthritic diathesis were 3 cases of uveitis and 1 of bilateral scleritis. In the patient who had scleritis, cortisone had a prompt and definite suppressive action on the ocular symptoms as well as on the manifestations of arthritis. Recurrence of the ocular disease was the rule when the use of cortisone was stopped, although final suppression of symptoms occurred after 3.8 Gm. of cortisone had been administered in 38 injections over an observation period of approximately four months. At the time of this report this patient had gone three months without recurrence of the scleritis.

Among the 3 patients with uveitis, cortisone had a beneficial effect on 2 and no effect on the third. In the latter case the uveitis was far advanced and had produced heavy posterior synechiae, keratic precipitates, and a complicated cataract in the affected eye. Of the 2 showing a favorable response to cortisone, 1 was still under investigation and the other had gone six months before experiencing a relapse of the uveitis. In 1 of these patients intra-ocular surgery was performed for the relief of secondary glaucoma during the phase of active uveitis. Cortisone so suppressed the symptoms of uveitis that the ocular surgery did not produce an expected exacerbation of the inflammatory process.

Of the 2 patients who seemed to have no association of the ocular disease with arthritis, 1 with advanced choroiditis seemed to derive no benefit from cortisone. In the other, uveitis that had failed to respond to many other therapies finally was brought under control by cortisone.

In the cases of vernal conjunctivitis, cortisone was found to have a definite suppressive effect on the subjective and objective symptoms of the disease, although the

symptoms recurred within a few days after the hormone was withdrawn.

Throughout this investigation period cortisone was used as a suspension of its acetate salt and was administered intramuscularly. In adult persons, 100 mg. usually was given daily. In children, 50 to 75 mg. was administered daily.

Details of the studies made during this seven month period will be published in a subsequent report.—*Author's abstract.*

Effects of Nitrogen Mustard on the Intraocular Pressure. HUGH DAVSON, ALFRED HUBER AND JUDD STREET. London, England. Brit. M. J. *2*:939-40, April 22, 1950.

Nitrogen mustard hydrochloride, when instilled into the conjunctival sac of rabbits, causes an acute hypertensive iritis with intra-ocular pressures rising to approximately 70 mm. of mercury.—*Author's abstract.*

Studies in Experimental Ocular Tuberculosis. XIII. Effect of Streptomycin and Promizole in Experimental Ocular Tuberculosis in the Normal Rabbit. ALAN WOODS, R. WOOD, AND H. NAQUIN. Baltimore, Md. Arch Ophth. *43*:834-48, May, 1950.

The effect of streptomycin and promizole on experimental ocular tuberculosis in the normal rabbit is reported. Under the experimental conditions there was complete control of visible inflammatory activity in all 15 animals. During the post-treatment observation period of 104 days there were visible inflammatory recurrences in only 3 rabbits. At the time the animals were killed 9 rabbits (60 per cent) showed either bacteriologic or histologic evidence of active ocular tuberculosis, whereas 6 (40 per cent) showed evidence, either bacteriologic or histologic, of complete cure. 2 references, 5 figures, 1 table.—*Author's abstract.*

BOOK REVIEWS

Eyes and Industry. By HEDWIG S. KUHN. St. Louis, C. V. Mosby Co., 1950. Ed. 2, 378 pages. Price: $8.50.

The second edition of *Eyes and Industry* by Dr. Kuhn is a comprehensive work including visual testing in industry, visual skills, industrial eye injuries, eye protection, illumination, and infections. The book is well written and illustrated by one who has had great experience in industrial ophthalmology, and her association with the Department of Industrial Psychology at Purdue University has afforded her extensive opportunity for research. A special chapter by Albert C. Snell, M.D., presents the latest facts on the medical and surgical care of eye injuries.

These days, every ophthalmologist is practicing with some relationship to industry, and therefore this book should be included in his library for study and reference. —*Truman L. Boyes, M.D.*

MISCELLANEOUS

Relationship of Pre-Optometry College Work to Performance in Optometry College Courses. M. H. BROWN AND H. W. HOFSTETTER. Los Angeles, Calif. Am. J. Optom. & Arch. Am. Acad. Optom. *27*:448-56, Sept., 1950.

This analysis represents a survey of the rated pre-admission and postadmission

performances of the members of a class of 89 students who graduated from the Los Angeles College of Optometry in 1949. The 89 students had taken their pre-optometry college work of not less than two years at 53 different institutions. Positive correlations were found to be of a significant value to use as a basis for prediction of performance in optometry college. The correlation between total pre-optometry performance and total postadmission performance was 0.586. The correlation of first semester optometry performance with performance in all subsequent semesters combined was 0.840. Other combinations of the data showed comparable correlation coefficients. The results of the present sample indicate that the grades earned in pre-optometry courses present a reliable basis for prediction of performance in optometry courses, even though the supply of applicants may arise from a variety of accredited institutions of higher education. The results did not indicate any justification for individual allowances to applicants on the basis of the size or geographic location of the accredited institution at which pre-optometric training was obtained. However there is evidence favoring the student with an excess quantity of pre-optometric education, even with the same rated quality of performance. Relationship of academic performance to professional success is briefly discussed.—*Author's abstract.*

Changes in Vision During Adolescence. ALBERT E. SLOANE AND J. ROSWELL GALLAGHER. Am. J. Ophth. 33:1538-43, Oct., 1950.

This report compares the findings at the initial visual screening examination, using a modification of the Massachusetts Vision Test of 228 boys (average age, 14½ years), with the findings on the same boys three years later. No change in the naked visual acuity of both eyes was found in 74.5 per cent of the group. There was a decrease of more than "one-line" in 7.5 per cent. There was no significant change in the results of the tests for heterophoria over the three year period.

The authors conclude that, although the frequency and extent of the changes in visual acuity observed in this group over a three year period are not great, nevertheless, they are sufficient to suggest the desirability of an annual visual screening examination of adolescents.—*Robert E. Bannon.*

The Peoria Visual Screening Project. CLIFTON S. TURNER AND J. A. POTTER. Illinois M. J. 97, March, 1950.

Initiated in 1948 for the purpose of determining the number of school children with visual difficulties and what could be done about them, the Peoria Visual Screening Project examined a total of 12,667 grade school students with the Massachusetts Vision Test.

The results indicated that approximately 18 per cent were below standard visually. The findings showed a gradual increase from 25 per cent in the first grade to 53 per cent in the eighth grade with visual difficulties. This indicates that visual problems increase as the child advances in school.

It is interesting to note that of those found to be below minimum visual standards, only 21 per cent obtained professional care. The authors stress the importance of a

small advisory committee representing both the professions of ophthalmology and optometry.

In order to overcome the lack of follow-up of unresponsive parents and to urge them to obtain proper visual care for their children, it is felt that this part of the program would be helped by assigning the follow-up work to public health nurses. —*Robert E. Bannon.*

Disturbances of Vision in Mental Disease. JAMES B. FUNKHOUSER. Richmond, Va. Am. J. Optom. & Arch. Am. Acad. Optom. *27*:484-90, Oct., 1950.

Eye symptomatology is frequently the concern of the neuropsychiatrist as well as the optometrist. But in many instances mentally sick patients will consult first with the optometrist when a visual complaint arises. The recognition of psychologic disorders of vision is a matter of some importance to those treating disease of the eye. Roughly, the eye complaints of mentally sick patients fall into three sub-groups: (1) the neuroses, (2) the psychoses, and (3) psychosomatic disease. Undue concern with trivial defect is usually neurotic. Bizarre symptomatology should lead the optometrist to suspect the possibility of a more severe mental disorder, particularly where the patient believes that his eyes have changed in color, form, or outward appearance, and that this change is apparent to others; or when patients believe that their eyes transmit or receive an evil influence (evidence that their primitive belief in the "evil eye" still exists in the subconscious of civilized patients). Further, there is much psychiatric evidence to suggest that emotional disorders can predispose to physical disease of the eye, such as hordeolum and other infections, as well as hysterical blindness, blepharospasm, squint, etc. —*Author's abstract.*

"BUT DOCTOR– I *CAN'T* WEAR THEM!

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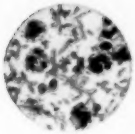

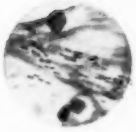

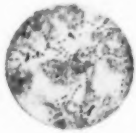

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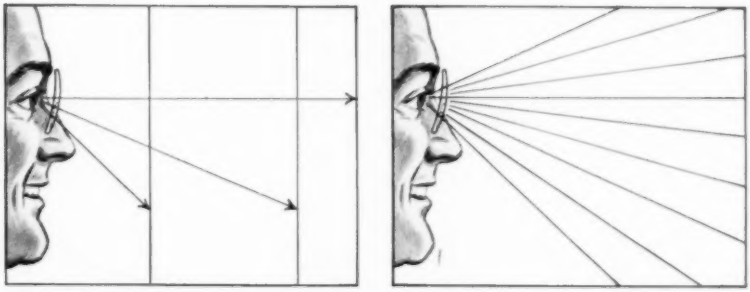

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